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PHYSICAL DIAGNOSIS

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PHYSICAL DIAGNOSIS

BY

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THIRD EDITION

THREE HUNDRED NINETEEN ILLUSTRATIONS

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MORGAN SMITH, M.D., LL.D.

DEAN OF THE MEDICAL DEPARTMENT
OF THE
UNIVERSITY OF ARKANSAS

PREFACE TO THIRD EDITION

In the preparation of this revision the section dealing wit physical examination of the circulatory system has been entirel rewritten and additional material incorporated to cover recen advances in the subject. Similarly, the chapter dealing wit blood pressure has been rewritten in its entirety. New illustr tions have been added to the text in several places, and old illustrations have been eliminated.

Polygraphy has been treated with greater detail in this ed tion; and a chapter dealing with electrocardiography and th diagnosis of the cardiac arrhythmias has been contributed b Dr. Drew Luten, Instructor in Clinical Medicine, Washingto University School of Medicine.

Minor changes in the text have been made in the chapters dea ing with bronchial asthma, diseases of the myocardium, an examination of the stomach.

W. D. R.

Little Rock, Ark.

PREFACE TO SECOND EDITION

The exhaustion of the present edition and the persistent d mand for the book are the incentives for this revision, in t preparation of which the author has availed himself of the su gestions contained in the kindly criticisms of the reviewers the original edition of the work.

In the preparation of the present volume the text has bee largely rewritten and supplemented by new material in order cover the recent advances in the subject. The clinical anatom of the various thoracic and abdominal organs has been carried step further in this edition, and the intimate correlation anatomy, pathology, and physical signs has been emphasize throughout the text. Additional space has likewise been allotte in the present edition to the physical principles underlying th various physical signs which are generated within the thorax and abdomen in health and in disease.

In the present edition, many of the borrowed illustrations of the first edition have been replaced by original drawings.

The chapter dealing with the x-ray as an aid in diagnosis has been revised and amplified by Dr. Dudley E. Mackey, of New York, formerly Instructor in Roentgenology, Cornell School of Military Roentgenology.

W. D. R.

Little Rock, Ark.

PREFACE TO FIRST EDITION

In the preparation of this volume the author has had in mind the medical student and the busy practitioner, and it has been his purpose to incorporate in a brief work the principles of physical diagnosis, together with the physical findings in the commoner diseases of the respiratory and circulatory systems. In this connection anatomy and pathology have been considered from the clinical standpoint, emphasis being laid upon these subjects as they influence the physical manifestations of disease of the thorax and abdomen.

In addition to the physical examination of the thoracic and abdominal viscera, it has seemed proper and practical to include in the work the principal diagnostic signs referable to the head, neck, and limbs, together with a minimum examination of the nervous system.

The work has been profusely illustrated, in the belief that free illustration is the nearest approach to personal contact in the teaching clinic.

The author wishes to express his appreciation to Dr. C. E. Shinkle, whose diagnostic table on the Bárány Tests is reproduced in the volume, for valuable assistance in preparing the section dealing with these tests. Many illustrations have been taken from other books, all of which have been credited in the text. He also wishes to thank Mrs. T. W. Marks for assistance rendered in the preparation of original drawings for the text; and the publishers for many courtesies during the preparation and publication of the volume.

W. D. Rose.

Little Rock, Ark.

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PHYSICAL DIAGNOSIS

PART I. THE THORAX

SECTION I

CHAPTER I

CLINICAL ANATOMY OF THE THORAX

The bony thorax resembles in shape a truncated cone with an anteroposterior flattening, which causes the transverse diameter to exceed the anteroposterior diameter by one-fourth. When the thorax is viewed in the living subject, however, the upper portion which corresponds to the apex of the cone which is formed by the bony thorax, exceeds in transverse diameter the lower portion of the thorax as a consequence of the investment of the thoracic cage by the bulky muscles of the shoulder girdle. The thoracic walls are formed by the rigid vertebral column posteriorly, and the ribs posteriorly, laterally, and anteriorly. The sternum enters into the formation of a large portion of the anterior wall; and posteriorly the thorax is reinforced by the overlying scapulæ, which cover the ribs upon either side of the vertebral column from the spinous processes of the second to the seventh dorsal vertebræ.

The intercostal spaces are occupied by the intercostal muscles which are further reinforced by the investing fascia and the parietal pleura. These spaces are traversed by the intercostal vessels and nerves, the latter sending out their lateral cutaneous branches in series at points situated midway between the spines of the thoracic vertebræ and the sternum. Similarly, the terminal filaments of the intercostal nerves emerge at points in series near the lateral margins of the sternum. In the presence of intercostal neuralgia the points of exit of these cutaneous nerves are not infrequently subject to pain upon pressure, constituting Valleix's points of tenderness.

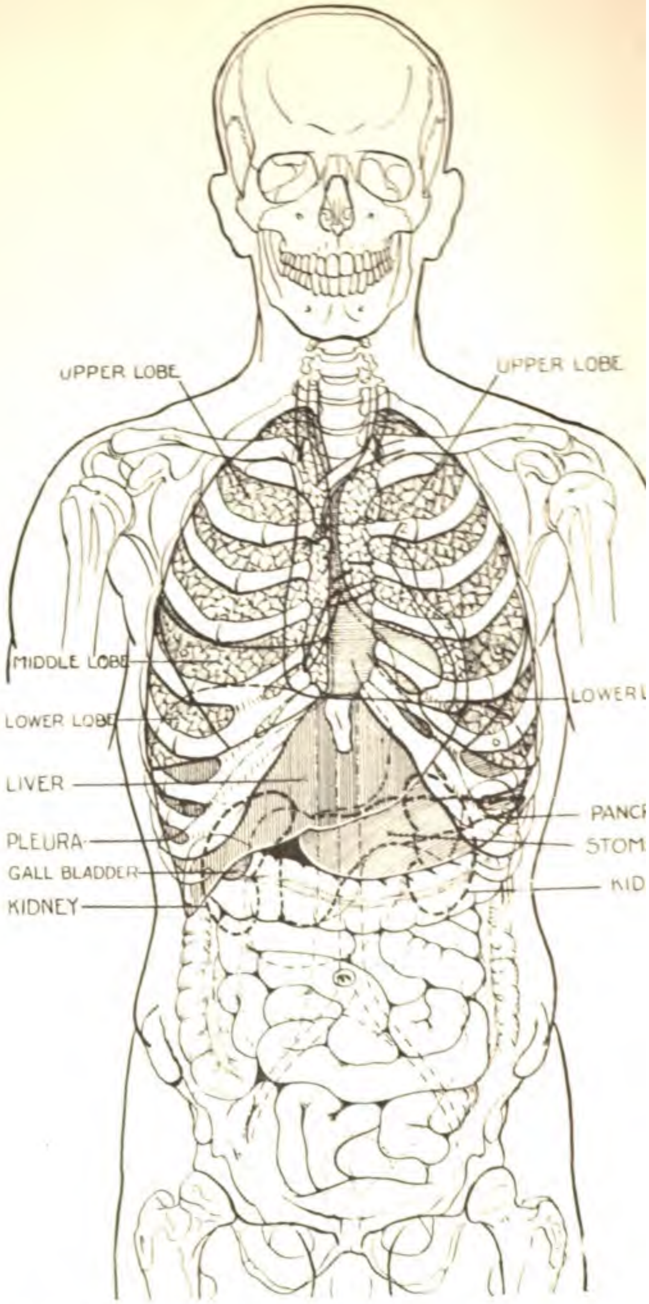

Fig. 1.—Relations and surface markings of thoracic and abdominal v
(Anterior view.)

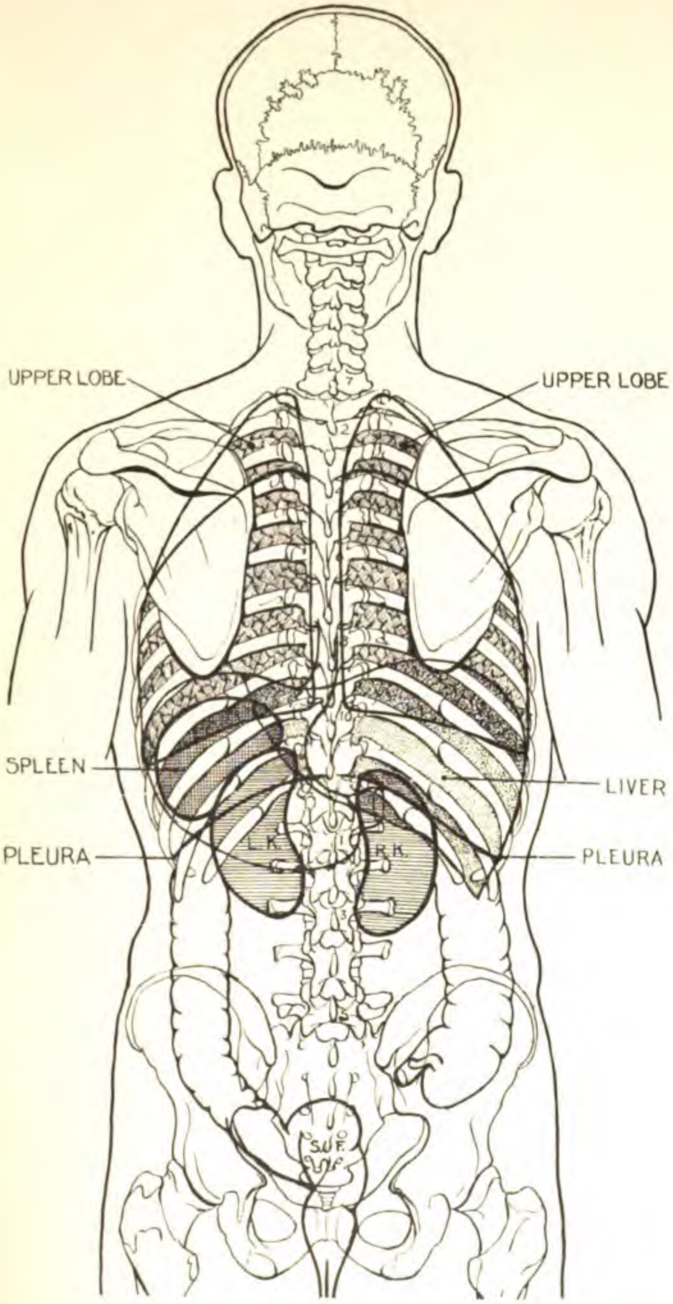

Fig 2.—Relations and surface markings of thoracic and abdominal viscera. (Posterior view.)

The *thoracic wall* varies in thickness in different regions of the chest, and also with the degree of physical development of the individual. Clothed in all parts by the overlying musculature, it is further reinforced posteriorly by the interposition of the osseous scapulæ, and anteriorly by the mammary gland in the female subject. In the axillary, infraaxillary, interscapular, and subscapular regions, on the other hand, the thoracic wall does not attain great thickness; consequently in these regions physical signs arising within the thorax are more clearly transmitted to the examining ear. In the subject of excessive muscular development as well as in the presence of general obesity a thick chest wall is the rule, while in the poorly nourished individual with imperfect development of the muscular system, the walls of the thorax are unduly thin and the scapulæ are readily displaced from their normal beds. The thoracic wall possesses less thickness in the female subject than in the male, owing to the greater degree of muscular development in the latter class. In the child, again, the chest wall is very thin and resilient, and physical signs arising within the thorax are transmitted thence with a corresponding increase of intensity.

The thoracic wall of the normal subject possesses an inherent elasticity, yielding readily to variations of pressure exerted from without or from within the cavity of the thorax. Consequently, characteristic deformity of the contour of the thorax readily results from occupation or from disease as, for example, the funnel-breast deformity of the shoemaker or carpenter, the barrel-chest of hypertrophic emphysema, or the elongated, alar thorax of chronic phthisis. As a result of this great plasticity of the thorracic parietes, careful inspection of the contour of the thorax assumes a very important rôle in physical diagnosis.

The *thoracic cavity*, containing the great organs of respiration and circulation, is limited inferiorly by the diaphragm and superiorly by the dome of the parietal pleura, which is reinforced in this region by a thickening of the investing fascia, Sibson's fascia. The capacity of the thoracic cavity is not commensurate with the external dimensions of the thorax. Inferiorly the dome of the diaphragm ascends as high as the upper border of the fifth rib in the right midclavicular line, and to the lower border of the fifth rib in the corresponding line upon the opposite half of the thorax; but, as if to compensate in some degree for this deficiency inferiorly, the cavity ascends beneath the dome of the pleura into the root of the neck to a height varying from one to one and one-half inches above the clavicle.

The thoracic cavity is divided by reflections of the parietal pleura into three chambers; namely, a *pleural cavity* upon either side of the median line, intervening between which is a third cavity, the *mediastinum.*

The *pleural cavities,* occupying the lateral regions of the thoracic cavity, extend upward into the base of the neck to the extent of one to one and one-half inches above the clavicle, and downward as low as the attachment of the diaphragm to the thoracic walls. The vertical diameter of each pleural cavity is diminished by the ascent of the diaphragm during expiration, whereas the transverse and anteroposterior diameters of the cavity are increased by the excursion of the chest wall during inspiration.

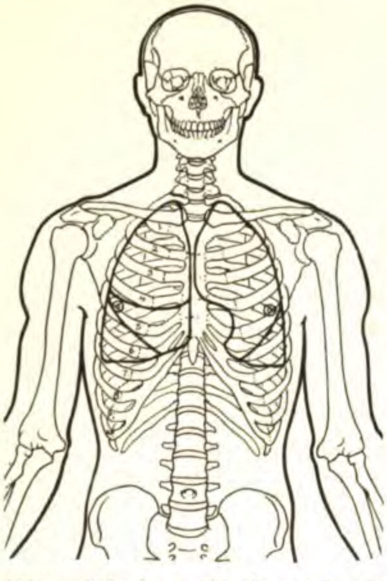

Fig 3.—Relations of the lungs with the anterior thoracic wall.

The pleural cavity is lined internally by a thin serous membrane, the pleura, which closely invests its walls, in which situation it is termed the *parietal pleura.* For purposes of description and accurate localization, several subdivisions of the parietal pleura are recognized according to the distribution of the membrane. The portion of the membrane which invests the anterior, lateral, and posterior walls of the cavity constitutes the *costal pleura;* the portion which clothes the superior surface of the diaphragm, forming the inferior limit of the pleural cavity, is designated the *diaphragmatic pleura;* while the folds which are reflected from the anterior to the posterior thoracic walls, forming in this wise the lateral walls of the mediastinum, are termed the *mediastinal pleura.* The same membrane is reflected from the mediastinal wall on to the lung at its root, clothing the external surface of the lung completely and dipping into the fissures of the organ, forming the *visceral pleura.*

In the normal subject the pleural membrane is moistened with a small amount of serous fluid, which permits the visceral and the parietal pleura to glide noiselessly over each other during the movements of the lung and the chest wall during respiration. In the presence of inflammation of the membrane, however, the pleural surface loses its smooth, glistening appearance, and becomes coated with a variable amount of fibrinous exudate, giving rise to a coarse or grating sound during the respiratory movements, which is recognized by the clinician as the pleural friction sound.

The *mediastinum,* the central portion of the thoracic cavity intervening between the pleural cavities, is limited anteriorly by the sternum and costal cartilages, and posteriorly by the bodies of the thoracic vertebræ, while its lateral walls are formed by the reflection of the parietal pleura from the anterior to the posterior wall of the thorax. This important portion of the thoracic cavity is arbitrarily divided into four subdivisions; namely, the superior, posterior, anterior, and middle mediastina.

The *superior mediastinum* is represented by the portion of the mediastinal space which is situated above the lower border of the manubrium sterni anteriorly and the lower border of the body of the fourth dorsal vertebra posteriorly. As in the case of the remaining subdivisions of the mediastinum, its lateral walls are formed by the mediastinal reflections of the parietal pleura. This space contains the intrathoracic portion of the trachea together with the tracheal glands, the esophagus, and the superior portion of the thoracic duct. The superior mediastinum also lodges the arch of the aorta, with the initial portions of the three great arterial trunks which spring from its convexity; and here also are found the innominate veins, uniting to form by their confluence the superior vena cava.

The *posterior mediastinum* represents the downward continuation of the posterior portion of the superior mediastinum, whence it extends as low as the upper surface of the diaphragm, which separates it from the abdominal cavity. Bounded laterally by the mediastinal pleura and posteriorly by the bodies of the dorsal vertebræ, the posterior mediastinum is in direct relation anteriorly with the pericardium and heart. The posterior mediastinum contains the descending thoracic aorta, the esophagus in close relation with the pneumogastric nerves, a portion of the thoracic duct and likewise a portion of the azygos veins. The space also lodges a chain of lymphatic glands, which are apt to become enlarged in the presence of intrathoracic malignant disease.

The *middle mediastinum* is situated immediately below the anterior portion of the superior mediastinum, and it is interposed between the anterior mediastinum ventrally and the posterior mediastinum dorsally, while its inferior limit or floor is formed by the central tendon of the diaphragm. That this is the most important subdivision of the mediastinum to the student of physical diagnosis is evident, when it is recalled that this limited space contains the pericardium and heart, the ascending aorta, the pulmonary artery with its two primary branches, the bifurcation of the trachea, the primary bronchi with their related bronchial glands, as well as the lower portion of the superior vena cava and its junction with the azygos veins.

The *anterior mediastinum* is a narrow space situated immediately behind the sternum. It is limited posteriorly by the pericardium and laterally by reflections of the parietal pleura from the anterior thoracic wall. The space is of little diagnostic interest, save tha it lodges a few lymphatic glands, which may become enlarged or become the seat of malignant disease.

As the mediastinum is traversed by such a variety of importan structures, it is evident that physical signs arising from mediastinal disease are apt in many instances to manifest themselves in the form of pressure symptoms. Pressure generated within thi space, being exerted upon the large blood vessels, upon the large lymphatic tract represented by the thoracic duct, upon the esophagus or upon the air passages, will in each instance produce physica signs of definite localizing value. Moreover, as a result of the close contiguity of so many vital anatomic structures within a restricted space, inflammation of a single structure is apt to involve adjacen organs, resulting in the formation of inflammatory adhesions o frank abscess formation.

The *thoracic viscera* comprise the air passages, represented by the larynx, trachea, and bronchi; the essential organs of respiration, the lungs; and the circulatory organs, represented by th pericardium and heart with the great vessels arising from its base

The *larynx* must be considered in conjunction with the trachea and bronchi, as it constitutes an essential portion of the air passages. This important structure occupies a position in the neck between the base of the tongue and the trachea, and opposite the bodies of the fourth, fifth, and sixth cervical vertebræ. In the ventral portion of the neck the larynx forms a visible prominence, the pomum Adami, lateral displacement of which is often significant of intrathoracic disease. Similarly, abnormalities in the respiratory movements of the larynx are often the deciding factor in the determination of the cause of dyspnea; while a palpable tug of the larynx is not infrequently symptomatic of aortic aneurysm.

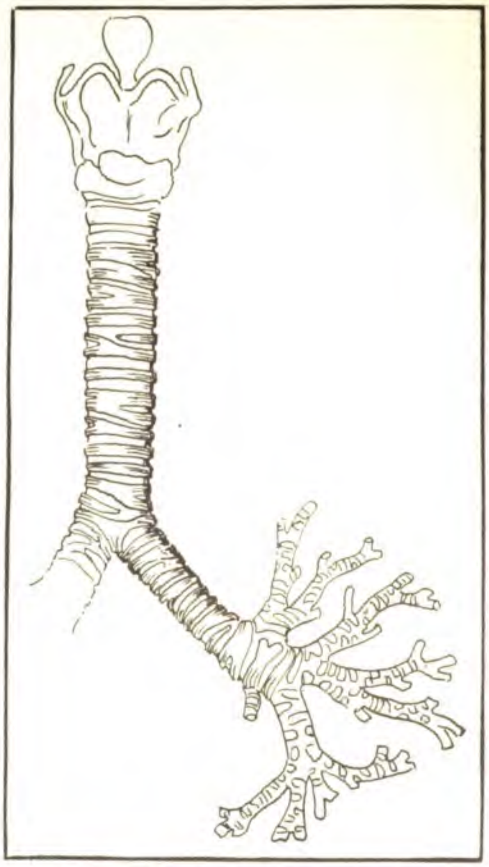

Fig. 4.—The bronchial tree. The walls of the bronchi contain cartilage in incomplete rings or plates distributed about their entire circumference. The cartilage and the elastic tissue make the tubes firm-walled; only the fine branches of one mm. or less in diameter have no cartilage, and are consequently collapsible. (From Brown.)

The cavity of the larynx contains the true and false vocal cords, intervening between which is the ventricle. The slight interval between the true cords constitutes the chink of the glottis or rima glottidis, variations in the dimensions of which influence the intensity of physical signs which are dependent upon the spoken voice for their production.

The mucous lining of the upper portion of the larynx is quite thick and vascular, and, in the presence of inflammatory affections of neighboring structures, is apt to swell from effusion into the submucous coat with the production of edema of the glottis, and stridulous respiration. The close adhesion of the mucous membrane to the true vocal cords prevents the effusion from extending below these structures.

The *trachea,* the second portion of the respiratory passage, has the form of a cylindrical tube with slight flattening of the posterior wall. Extending from its junction with the cricoid cartilage of the larynx opposite the sixth cervical vertebra to its bifurcation to form two primary bronchi at the level of the fourth dorsal vertebra, in the neck it is covered by the flat infrahyoid muscles, the isthmus of the thyroid gland, and the integument; while within the thorax it is covered by the thymus gland in the child, and by the remnant of this gland and by the arch of the aorta in the adult subject. The trachea of the adult is from four to four and one-half inches in length.

The trachea is a composite structure formed by from sixteen to twenty incomplete cartilaginous rings, invested externally by a fibrous membrane and lined internally by a mucous membrane containing numerous mucous glands, whose secretion prevents excessive dryness of the sensitive mucous lining of the tube. In the presence of tracheobronchial irritation and inflammation, these glands pour out an excessive, tenaceous secretion.

The free ends of the cartilaginous rings, of which the trachea is largely composed, are directed posteriorly, in which situation the deficiency in the wall of the tube is closed by transverse bundles of involuntary muscle, constituting the trachealis muscle of Todd and Bowman. The transverse bands of this muscle, the contraction of which serve to diminish the lumen of the trachea, are invested externally by bands of nonstriated muscular fibers which extend the entire length of the tube.

The *bronchi* pass obliquely downward and outward from their origin at the tracheal bifurcation to enter the roots of their respective lungs. The *left bronchus,* which is approximately two inches in length, enters the root of the left lung at a point opposite the sixth thoracic vertebra. In its course the left bronchus crosses the esophagus, thoracic duct, and descending aorta. The left pulmonary artery occupies a position at first superior to, and in its course finally comes to occupy a position posterior to, the bronchus. The arch of the aorta crosses above the left bronchus; and, in the presence of aneurysm of this portion of the vessel, is apt to produce a downward displacement of the bronchus which is responsible for the systolic trachael tug which not infrequently characterizes this condition. As the left bronchus is smaller and more deeply situated in the thoracic cavity than is the corresponding bronchus of the right side, and as it forms a more acute angle with the trachea than does the right bronchus, physical signs arising within it are not conducted to the surface of the thorax with the same degree of intensity as are similar sounds generated within the right bronchus.

The *right bronchus*, approximately one inch in length, passes almost vertically downward to enter the root of the right lung opposite the fifth thoracic vertebra. The vena azygos major courses upward behind the right bronchus; and, arching over the tube, joins the superior vena cava, which is in relation with the bronchus anteriorly. The right pulmonary artery occupies initially a position below the corresponding bronchus, and in its further course occupies a position anterior to it.

The *tracheobronchial lymph nodes* are collected into four principal groups along the course of the trachea and bronchi. A group of these glands occupies the angle between the trachea and right bronchus upon the right side, while a similar group occupies the angle between the trachea and the left bronchus upon the opposite side. A large group of glands is clustered in the angle which is formed by the tracheal bifurcation. Finally, the interbronchial nodes occupy the angles of bifurcation of the larger bronchi throughout the pulmonary parenchyma. With advancing age these glands become deeply pigmented as a result of the deposition of carbonaceous material from the pulmonary alveoli and finer bronchioles; and in the presence of malignant disease they become enlarged with the production of pressure signs.

The *lungs*, suspended by their respective roots, and covered upon their surfaces by the visceral pleura, hang free within the pleural cavities. The lungs during life entirely fill the pleural sacs, so that the latter represent only potential cavities. Each lung possesses an apex, which ascends one to one and one-half inches above the clavicle beneath the dome of the pleura; a base, which rests upon the convex superior surface of the diaphragm; a convex or costal surface, which is in contact with the anterior lateral, and posterior thoracic walls; and an irregular internal surface, which bears the imprint of a number of structures contained within the mediastinum with which it is in relation.

The *left lung* is divided into two lobes by a deep fissure, which extends well in toward the root of the lung, and which is lined by a reduplication of the visceral pleura. In inflammation of the pleura the portion of the membrane which dips into the fissure may be the only portion of the membrane involved, with the consequent production of a condition of interlobar pleurisy very difficult of detection by physical means.

The upper lobe of the left lung comprises a large portion of the external surface and the entire anterior border of the lung, while the lower lobe comprises the entire base and the greater portion of the posterior border of the lung. This is an anatomic fact of considerable importance, as during a physical examination it is frequently desirable to ascertain whether a morbid process having its inception in the apex or upper lobe of the lung has progressed to the lower lobe.

The *right lung* is divided into three lobes by two fissures. The upper lobe comprises the apex, a little more than half of the external surface, and the portion of the anterior border of the lung above the level of the fourth costal cartilage. The lower lobe comprises the entire base of the lung, but only a limited portion of the external surface. The middle lobe of the right lung is a wedge-shaped portion interposed between the upper and lower lobes, comprising the anterior portion of the external surface of the lung below the level of the fourth costal cartilage.

The external surface of each lung is convex; whereas the internal surface, which is in contact with the mediastinum, presents depressions corresponding to the mediastinal structures with which it is in relation.

The internal surface of each lung is marked by a rather deep depression, which receives the pericardium and heart. This depression is much more distinctly marked upon the left lung, owing to the projection of the heart to the left side of the median line of the thoracic cavity. Situated immediately above and behind the cardiac depression, each lung presents the hilus or pulmonary root, for the entrance of the primary bronchus with the accompanying vessels, lymphatics, and nerves of the lung; while extending downward from the hilus is a fold of the reflected pleura, the ligamentum latum pulmonis.

In addition to these impressions, the internal surface of the left lung is traversed by a fairly deep groove, which curves above the left bronchus and descends behind this tube, and which lodges the aorta. A second groove, which lodges the subclavian artery, passes upward from the aortic groove at the point where the latter arches over the pulmonary root.

The internal surface of the right lung is traversed by a groove which, arching over the right bronchus, lodges the vena azygos major. Extending upward from the fore part of this groove is a second groove which is traversed by the superior vena cava.

The lungs present certain individual differences which have an influence upon physical signs emanating from the two sides of the thorax. The apex of the right lung mounts to a greater height in the root of the neck beneath the dome of the pleura than does that of the left lung. Similarly, the base of the right lung occupies a higher level than does the base of the left lung. But, in addition to its slightly higher position with reference to the opposite lung, the right lung has a greater transverse diameter than its fellow of the opposite side. Moreover, the anterior border of the right lung is approximately vertical, approaching the median line; whereas the corresponding border of the left lung falls away from the median line in an oblique direction, exposing a portion of the right ventricle and pericardium in the interval which is created in this manner.

The extent to which the anterior borders of the lungs approach each other varies with the depth of inspiration and the integrity of the pulmonary parenchyma, as well as with the state of the mediastinal structures. In the absence of mediastinal disease, upon forced inspiration the anterior pulmonary borders come forward and cover the base of the heart and the great vessels arising therefrom and enveloped by the pericardium. At the completion of inspiration the anterior borders of the lungs are in contact from the lower border of the manubrium sterni to a point corresponding to the level of the fourth costal cartilage. At this stage of the respiratory cycle the anterior border of the right lung is vertical and parallel with the median line of the thorax, while the obliquely directed anterior border of the left lung covers all of the pericardium and heart save a limited portion corresponding to the lower third of the right ventricle. During tranquil respiration the anterior borders of the lungs do not come into contact at any point. Owing to the projection of the heart toward the left of the median line, the anterior border of the right lung is the more mobile of the two, and during tranquil inspiration approaches nearly to the median line behind the sternum.

Various factors may, however, modify the excursions of the anterior pulmonary borders. In the presence of excessive cardiac hypertrophy or extensive pericardial effusion the borders do not meet over any portion of the pericardium. The same condition obtains in the absence of cardiac or pericardial disease when the heart is pushed forward by disease in the posterior mediastinum. Moreover, the excursion of the pulmonary borders is restricted by the formation of adhesions between visceral and parietal pleura, or by cirrhotic changes in the pulmonary parenchyma.

As the anterior border of the right lung enjoys a greater freedom of movement than does the corresponding border of its fellow, so also the lower border of the right lung expands more freely than does that of the left lung. The apex of the left lung, on the contrary, expands more freely than does that of the right lung. During both tranquil and forced respiration the posterior portions of the lungs remain quietly in relation with the walls of the pleural cavities, while the apices, anterior borders, and bases expand and recede with inspiration and expiration. The excursion in any of these directions is apt to become deficient as the result of traction from adhesions or retrogressive changes in the lung.

The internal structure of the lung is very intimately related to many symptoms and signs which are manifested during disease of these organs. The primary bronchus, which enters the lung at the hilus, divides dichotomously until, after repeated divisions, very fine branches termed bronchioles are formed. The bronchioles have no communicating branches with the result that when an obstruction of one branch develops, air is withheld from the vesicles in which it terminates, and a condition of localized atelectasis inevitably results.

In the bronchiole there is a gradual transition of the stratified columnar epithelium of the bronchus into simple columnar epithelium, which in turn, near the distal end of the terminal bronchiole, gives place to small groups or islands of flat epithelial cells, *respiratory epithelium*. The epithelial lining of the terminal bronchiole is supported by a thin basement membrane, beneath which is a tunic containing numerous elastic fibers and circularly disposed bands of involuntary muscular fibers, spasm of which possibly plays a part in the production of the paroxysm of bronchial asthma.

Each terminal bronchiole terminates in an irregularly pyramidal chamber, the *infundibulum*, which constitutes the "blood-vascular unit" of the lung. The walls of the infundibula comprise a series of minute, blind pouches, the *alveoli*, which are lined with a single layer of flat respiratory epithelium supported by a delicate basement membrane containing numerous elastic fibers. These elastic fibers render the infundibular walls very resilient and play an important part in the expansion and recession of the lung during respiration.

Each infundibulum is invested by a dense capillary plexus derived from branches of the pulmonary artery, which accompany the bronchi and bronchioles in their ramifications throughout the lung. The capillary plexus surrounding each infundibulum is disposed in a single layer, and there is no communication with the vessels of adjacent infundibula. The blood content of these capillaries is very intimately exposed to the air in the infundibula, as they are separated only by three very thin membranes; namely, the endothelium of the capillary wall, the delicate basement mem-

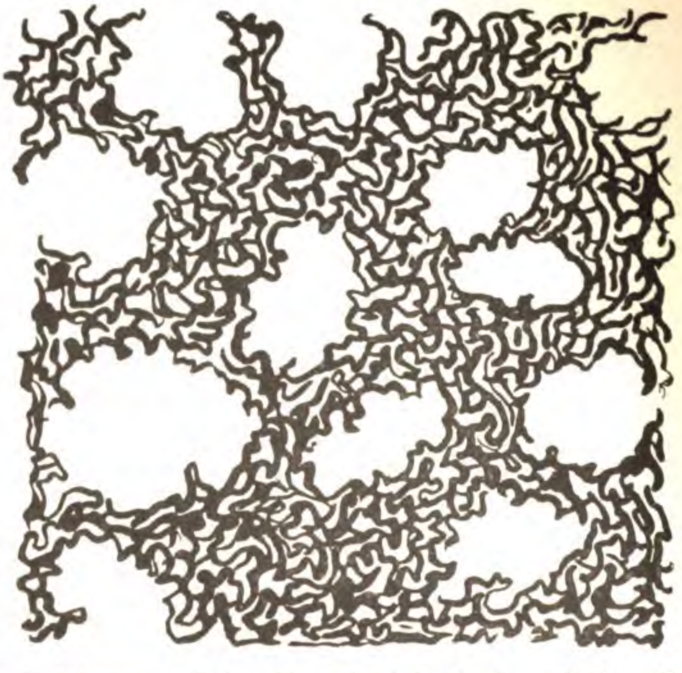

Fig. 5.—Pulmonary capillaries. The walls of the alveoli are thickly studded with capillaries; any marked alteration of alveolar air tension will therefore have a profound effect upon the circulation. (Brown, after Bohm, Davidoff, and Huber.)

brane of the infundibulum, and the single layer of infundibular epithelium. In hypertrophic emphysema, when the interalveolar septa are destroyed, as the capillary plexus of each infundibulum is distinct and has no communication with those of adjacent infundibula, the amount of blood exposed to the air in the large cavities which are formed by coalescence of several infundibula is considerably reduced, leading to dyspnea upon slight exertion in this class of patients.

The *lymphatics* of the lungs drain into the tracheo-bronchial lymph nodes and the mediastinal glands, with the result that these glands are early involved in tuberculous infection of the lungs, or when the lung is the seat of malignant disease. Similarly, these glands serve as filters for the irritant dusts which are conveyed to them by phagocytes from the finer bronchioles and infundibula in pneumonokoniosis.

Topographical Anatomy of the Thorax

The Pleura.—The surface markings of the pleura correspond to a line drawn from either sternoclavicular articulation downward and inward to the transverse ridge which marks the junction of the manubrium and gladiolus of the sternum. Thence the anterior borders of the reflections of the pleural membranes pass vertically downward slightly to the right of the median line to the level of the fifth intercostal space. At this point upon the surface of the thorax the two membranes separate, and in their further course diverge the one from the other.

The right pleura continues vertically downward almost to the junction of the gladiolus and the ensiform cartilage, whence it pursues an oblique course, passing outward, downward, and backward toward the vertebral column. In its course the lower border of the pleura crosses the seventh rib in the midclavicular line, the ninth rib in the midaxillary line, and the eleventh rib in the scapular line.

At the level of the fifth intercostal space the left pleura pursues a course toward the left and then downward to gain the posterior surface of the sixth costal cartilage in the left parasternal line. The membrane crosses the sixth costal cartilage vertically and from its lower border is reflected downward, outward, and backward toward the vertebral column, occupying a slightly lower level than does the lower border of the right pleura. In the interval which remains between the left sternal border and the left pleural membrane in the fifth intercostal space, an interval which corresponds accurately with the *incisura cardiaca* of the anterior border of the left lung, a limited portion of the right ventricle is brought into direct relation with the anterior thoracic wall.

The superior limit of the supraclavicular portion of the pleura, representing the dome of the pleural cavity, is indicated by a line drawn obliquely upward and outward from the sternoclavicular articulation upon either side, crossing the lower portion of the root of the neck so as to curve upward and descend to the spine of the seventh cervical vertebra. The maximum height of the curve, which corresponds to the apex or dome of the pleural cavity, is one to one and one-half inches above the clavicle.

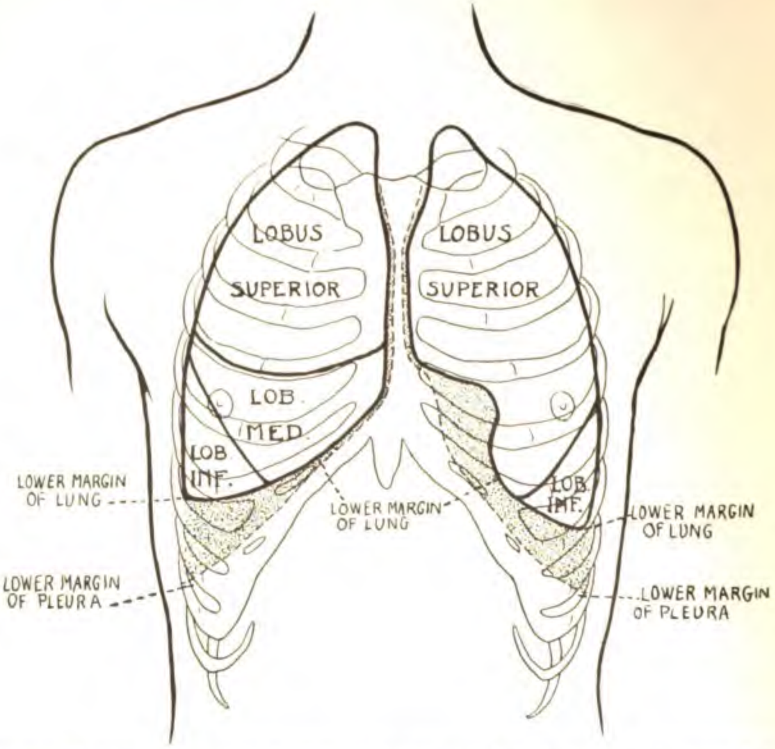

Fig 6.—Illustrating the normal borders of the lungs and the location of the interlobular septa. Anterior view. (Pottenger, after Corning.)

Upon the posterior surface of the thorax the course of the pleural reflection is represented by a vertical line drawn along either side of the vertebral column from the level of the seventh cervical vertebra to the articulation of the eleventh rib with the vertebral column, whence the line is continued downward and outward in a gentle curve to meet the line of reflection of the anterior portion of the membrane.

The Lungs.—The borders of the lungs correspond accurately with the line of reflection of the pleura, save that inferiorly the lower borders of the lungs fall short of the pleura by one intercostal space, being found at the level of the sixth rib in the midclavicular line, the eighth rib in the midaxillary line, and the tenth rib in the scapular line. The interval between the lower border of the lung and the inferior limit of the pleural reflection upon either side, representing one intercostal space upon the surface of the thorax, is the *complementary sinus,* into which the lower border of the lung descends during forced inspiration. These surface

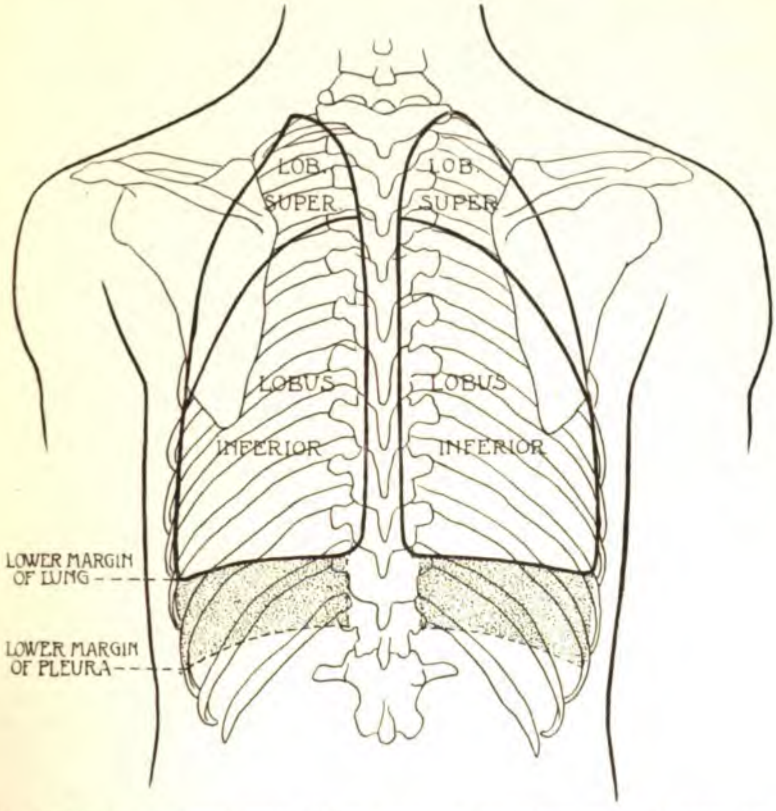

Fig. 7.—Illustrating normal borders of the lungs and interlobular septa. Posterior view. (Pottenger, after Corning.)

markings should be borne in mind when determining the total expansion of the lungs by percussion.

Fissures and Lobes of the Lungs.—The position of the great fissure, which is common to both lungs and which intervenes between the upper and lower lobes, is represented by a line drawn upon the surface of the thorax from the spinous process of the third thoracic vertebra obliquely downward and forward to the lower border of the sixth rib in the midclavicular line. The shorter fissure, intervening between the upper and middle lobes of the right lung is represented by a line drawn upon the surface of the thorax from the apex of the axilla almost horizontally forward to meet the sternum at the level of the fourth right costal cartilage.

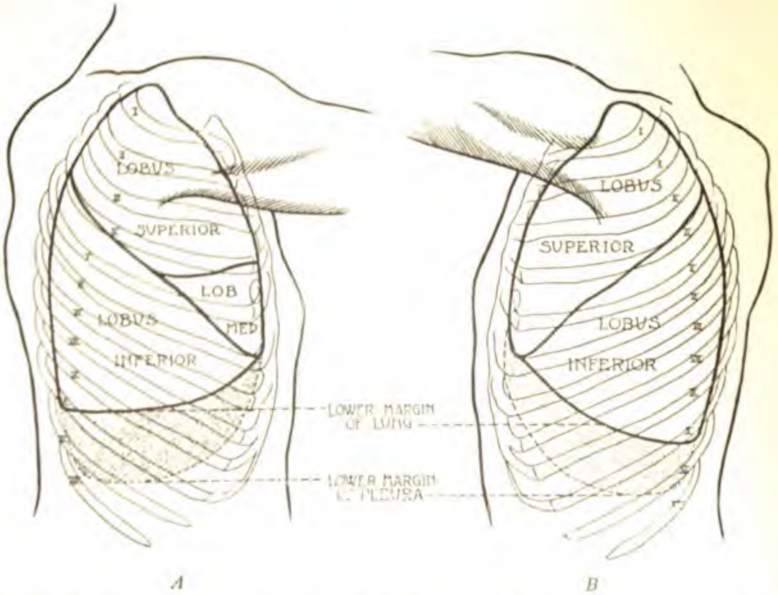

Fig. 8.—Illustrating the normal borders of the lungs and the location of the interlobular septa. Lateral view. *A*, right; *B*, left. (Pottenger, after Corning.)

Trachea and Bronchi.—The course of the trachea corresponds to a broad line passing vertically downward from the upper margin of the manubrium sterni to the level of the upper border of the second rib in the median line of the thorax. At this point upon the surface of the thorax the trachea divides to form the two primary bronchi, which diverge from each other in a downward and outward direction, the right bronchus inclining more directly downward than does the left.

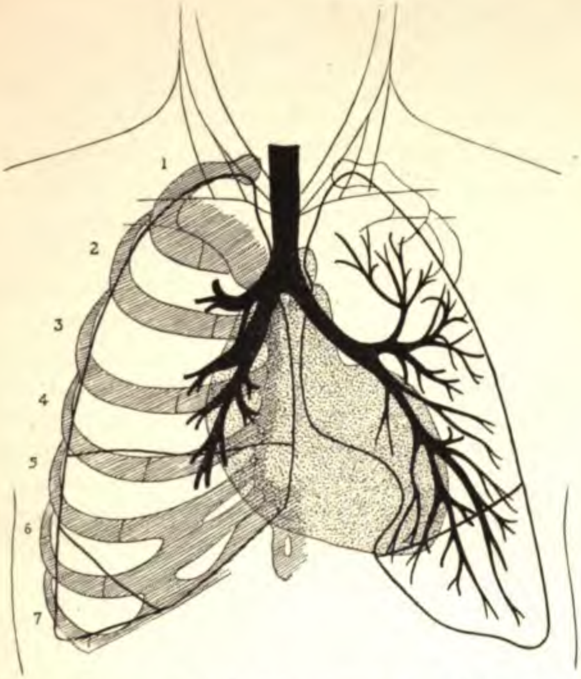

Fig. 9.—Showing the position of the bifurcation of the trachea and the peri-tracheal and peri-bronchial glands projected upon the anterior surface of the chest in a young adult. (Pottenger, after Gerhartz.)

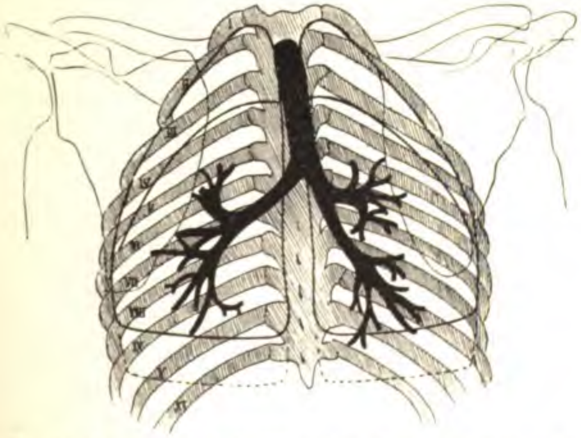

Fig. 10.—Showing the position of the bifurcation of the trachea with the peri-tracheal and peri-bronchial glands projected upon the posterior surface of the chest in a young adult. (Pottenger, after Piersol.)

intercostal space is named in accordance with the number of the rib immediately above it; hence the first intercostal space occupies the interval between the first and second ribs.

In *counting the ribs* one of several methods may be employed. In counting the ribs upon the anterior surface of the thorax it is convenient to commence the enumeration at the angle of Louis, which corresponds accurately to the second chondrosternal articulation. In counting the ribs upon the posterior surface of the thorax the inferior angle of the scapula may be utilized as a point of departure, as the tip of this bone overlies the seventh rib when the thorax is in repose. In the enumeration of the ribs upon the lateral aspect of the thorax the highest digitation of the serratus magnus muscle becomes the normal landmark, as this portion of the muscle overlies the sixth rib. The muscle may be rendered tense by abduction of the arm to a horizontal position. Certain clinicians prefer to proceed from below in counting the ribs, beginning with the extremity of the twelfth rib, which is palpable in the majority of subjects.

The *mammary gland* in the male subject is a rudimentary structure. In the female subject, however, it is well developed, and extends from the third to the seventh intercostal space in the midclavicular line. In the male subject the *nipple* is a reliable guide to the fourth intercostal space; but in the female, owing to the pendulous condition of the breast, it is an unreliable landmark.

The *scapula* overlies the dorsal aspect of the bony thorax, extending along the vertebral column from the second to the seventh rib. Always readily palpable, the bone stands out prominently in the form of the "winged scapula" of the alar thorax of chronic phthisis.

The *vertebral column*, or *spine*, in persons of excessive muscular development is represented by a linear, median furrow, and the spinous processes are palpated with difficulty. But in thin subjects and in children many of the spinous processes are visible with the patient in the erect posture; and upon bending the trunk forward they are readily palpated and counted. The spinous process of the seventh cervical vertebra is always a conspicuous landmark, and it may be employed as a point of departure in the enumeration of the vertebræ. Also the inferior angle of the scapula, which corresponds to the level of the spinous process of the seventh thoracic vertebra, may be utilized as a starting point in the enumeration. Lateral curvature of the vertebral column (scoliosis) or abnormal degrees of anteroposterior curvature (lordosis) may be encountered in connection with the thoracic deformities of rickets, hypertrophic emphysema, and in other forms of pulmonary disease.

Topographical Lines and Regions

For purposes of clinical description and for convenience in localizing lesions arising within the thorax, a number of regions may be outlined upon the surface of the thorax by means of arbitrary vertical and horizontal lines. It is of the first importance that the student should become thoroughly familiar with the relations of the thoracic viscera to these several regions.

Vertical Lines.—The *midsternal line* is a vertical line erected upon the anterior aspect of the thorax, traversing the midportion of the sternum from the midpoint of the superior border of the manubrium sterni to the tip of the ensiform cartilage.

The *sternal line* conforms to the lateral border of the sternum from the sternoclavicular articulation to the junction of the gladiolus with the ensiform cartilage, whence it is continued in a direction downward and outward along the course of the lower border of the costal arch to meet the anterior axillary line.

The *midclavicular line* is a vertical line dropped upon the anterior thoracic surface from the midpoint of the clavicle. It frequently, though not invariably, passes through the nipple and hence is frequently termed the *nipple line*, or the *mammary line.*

The *parasternal line* is a vertical line occupying a position upon the anterior aspect of the thorax midway between the sternal and the midclavicular lines.

The *anterior axillary line* is a vertical line dropped along the lateral thoracic wall from the anterior fold of the axilla.

The *midaxillary line* is a vertical line which passes down the lateral wall of the thorax from the apex of the axilla.

The *posterior axillary line* is a line which is dropped along the lateral thoracic wall from the posterior fold of the axilla.

The *scapular line* is a vertical line drawn upon the posterior surface of the thorax in such fashion as to pass through the tip of the inferior angle of the scapula.

The *midspinal line* is a vertical line which conforms to the series of spinous processes of the vertebræ.

Horizontal Lines.—The *cricoclavicular line* is drawn from the [illegible] of the cricoid cartilage outward and with a slight inclination downward to meet the prominence caused by the outer extremity of the clavicle.

The *clavicular line* is a horizontal line passing outward from the sternoclavicular articulation, conforming to the course of the clavicle.

The *third costal line* is drawn horizontally outward from the sternal line at the level of the third costal cartilage to meet the anterior axillary line.

The *sixth costal line* is a horizontal line commencing at the sternal line at the level of the sixth chondrosternal articulation, which, after crossing the anterior axillary and midaxillary lines, terminates at the posterior axillary line.

The *scapular spinal line* is a horizontal line projected outward upon the posterior thoracic wall from the scapular line, and conforming to the course of the spine of the scapula.

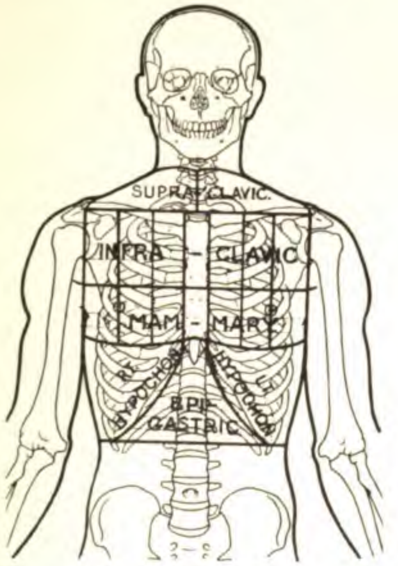

Fig. 11.—Topographic regions of the thorax. (Anterior view.)

The *infrascapular line* is a line drawn horizontally across the posterior surface of the thorax at the level of the inferior angles of the scapulæ.

The *twelfth dorsal line* is drawn from the point overlying the ous process of the last thoracic vertebra, passing downward and vard to meet the posterior axillary line.

rions of the Thorax.—Through the medium of these various l and horizontal lines, a number of regions are mapped out surface of the thorax.

The *sternal region* overlies the sternum and is bounded above by the episternal notch, below by the scrobiculus cordis, and laterally by the sternal lines. The upper portion of the sternal region, overlying the manubrium sterni, contains the anterior borders of the lungs, together with the bifurcation of the trachea, and the arch of the aorta. This region also is traversed by the left innominate vein and the beginning of the superior vena cava. The lower portion of the sternal region, corresponding to the distribution of the gladiolus, overlies the greater portion of the right ventricle together with the edge of the left ventricle, as well as a portion of the anterior borders of both lungs. Within this region also are found the origin of the pulmonary artery and the ascending aorta, and the space also includes the right auricle. This region overlies the attachment of the pericardium to the diaphragm, and also a limited portion of the left lobe of the liver.

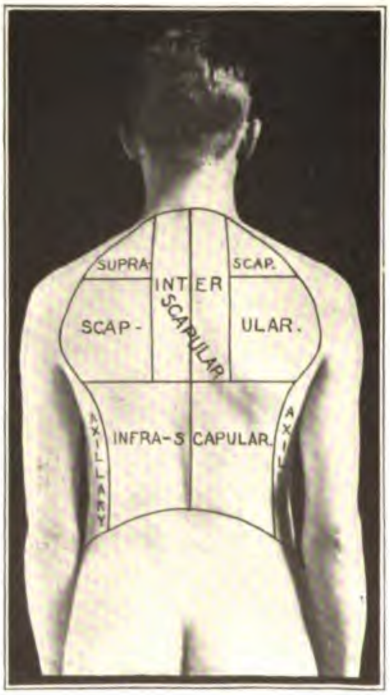

Fig. 12.—Topographic regions of the thorax. (Posterior view.)

The *supraclavicular region* lies in the base of the neck, above the clavicle, bounded above by the cricoclavicular line and inferiorly by the clavicular line. These regions, upon either side, contain the apices of the lungs, the common carotid and subclavian arteries, together with the subclavian and jugular veins.

The *infraclavicular region* is situated immediately below the clavicle. It is bounded above by the clavicular line, below by the third costal line, internally by the sternal line, and externally by the anterior axillary line. The anatomic structures lying within the limits of the infraclavicular regions differ upon the two sides of the thorax. The *left infraclavicular region* overlies the upper lobe of the left lung; and, adjacent to the left sternal border, a portion of the left auricle and the left pulmonary artery, as well as a portion of the left bronchus. The *right infraclavicular region* contains within its limits the greater portion of the upper lobe of the right lung, with the termination of the right bronchus, and the right pulmonary artery.

The *mammary region* lies immediately below the infraclavicular region, upon either side of the thorax, limited above by the third costal line, below by the sixth costal line, internally by the sternal line, and externally by the anterior axillary line.

As in the case of the infraclavicular regions, so also in the mammary regions the structures which are contained within the limits of these regions vary widely upon the two sides of the thorax. The *left mammary region* contains a portion of both lobes of the left lung, as well as the greater portion of the heart and pericardium, the latter partially overlapped by the anterior border of the left lung. The *right mammary region* contains a portion of the right auricle and right ventricle, partially overlapped by the anterior border of the right lung, which occupies the major portion of this region. The right lobe of the liver, clothed by the diaphragm, ascends in this region as high as the upper border of the fifth rib in the midclavicular line.

The *hypochondriac region* lies immediately below the mammary region, upon either side of the thorax, between the sixth costal line, the anterior axillary line, and the downward and outward continuation of the sternal line along the line of the costal arch. The contents of the regions vary upon the two sides of the thorax.

The *left hypochondriac region* contains the complementary sinus into which the lower border of the left lung descends during inspiration, the cardiac end of the stomach, the diaphragm, and the tip of the left lobe of the liver. The *right hypochondriac region*, in ition to the lower border of the right lung, contains within its ts the right lobe of the liver, covered by the diaphragm.

axillary region occupies the superior portion of the lateral aspect of the thorax, limited superiorly by the apex of the axilla, inferiorly by the sixth costal line, anteriorly by the anterior axillary line, and posteriorly by the posterior axillary line. This region upon both sides of the thorax contains only pulmonary tissue, hence upon percussion it yields a frankly resonant note.

The *infraaxillary region,* lying immediately below the axillary region upon the lateral aspect of the thorax, is bounded by the anterior and posterior axillary lines, the sixth costal line, and the downward prolongation of the sternal line along the costal arch.

The *left infraaxillary region* contains the lower portion of the left lung, the lower border of which reaches the eighth rib in the midaxillary line. The space also contains the diaphragm, and below this muscle a portion of the stomach and the spleen. The *right infraaxillary region* contains, in addition to the right lung and the diaphragm, a portion of the right lobe of the liver.

The *suprascapular region* overlies the supraspinous fossa of the scapula upon the posterior wall of the thorax, and is limited below by the scapular spinal line. The only structure within this region is the pulmonary apex upon either side of the thorax.

The *scapular region,* overlying the infraspinous fossa of the scapula, is limited superiorly by the scapular spinal line and inferiorly by the infrascapular line. This region upon either side of the thorax contains the posterior voluminous portions of the lungs, including a portion of both upper and lower lobes.

The *infrascapular region* upon either side of the thorax is limited superiorly by the infrascapular line, inferiorly by the twelfth dorsal line, and externally by the posterior axillary line. The anatomic structures within the limits of this region vary upon the two sides of the thorax.

The *left infrascapular region* overlies the lower lobe of the left lung, its inferior margin reaching the tenth rib in the scapular line. This region also contains the thoracic aorta in the lower portion of its course, and below the diaphragm a portion of the left kidney and of the spleen. The *right infrascapular region* contains, in addition to the lower lobe of the right lung, a portion of the right lobe of the liver, and of the right kidney.

The *interscapular region* occupies the interval upon either side of the thorax between the scapular line and the midspinal line, limited inferiorly by the infrascapular line. The structures contained in the two regions are different upon the two sides of the thorax.

The *left interscapular region,* in addition to the left lung, contains a portion of the left bronchus; and, near the midspinal line, the descending thoracic aorta, the esophagus, and the thoracic duct. The *right interscapular region* overlies the right lung and right bronchus in a portion of its course. The trachea extends downward in front of the bodies of the thoracic vertebræ from its junction with the larynx opposite the sixth cervical vertebra to the fourth dorsal vertebra, where it bifurcates to form the bronchi. The tracheobronchial glands are clustered about the angles formed by the bifurcation.

SECTION II

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS

CHAPTER II

INSPECTION

Object and Technic.—In the study and analysis of disease of the respiratory organs, inspection is employed to determine the condition of the surface of the thorax, various unilateral and bilateral variations from the normal contour and size of the thorax, the presence of local prominences and depressions, the character and frequency of the respiratory movements of the thorax, the relative and absolute degree of expansion of the two sides, the presence of local abnormal pulsations upon the surface of the thorax, and in fluoroscopic study of the thoracic viscera.

During inspection of the thorax the clothing should be removed to the waist, as a full and direct exposure of the chest is essential to a proper examination by all the methods employed in physical diagnosis. During the routine examination the erect posture, with the patient seated or standing, is preferable. During the inspection of the thorax, as in all physical examinations, the attitude of the patient should be natural and unconstrained. In the routine inspection of the front of the thorax the arms should hang naturally at the sides. During inspection of the lateral regions of the thorax the hands should be clasped behind the head, allowing a free exposure of the axillary and infraaxillary regions. In examining a patient in the erect posture, the examiner should view the chest from the front, from the sides, and from behind the patient. Finally, he should assume a position above the patient and look downward over the shoulders of the patient. In the event that it is necessary to examine a patient in the recumbent posture, the thorax should be inspected from below, the examiner assuming a position near the feet of the patient, as well as from above, the examiner standing near the head of the patient, with the patient in such posture as to allow a free exposure of the lateral and anterior regions of the thorax. Moreover, in such event, the patient must be turned a sufficient number of times during the examination to insure the inspection of all portions of the thorax. The element of position of the patient is one of the most important factors which enter into physical examination, as a faulty or constrained attitude upon the part of the patient is a fruitful source of erroneous conclusions upon the part of the examiner.

During the examination of the patient in the erect posture the light should, in the first instance, fall directly upon the area under examination, the source of illumination preferably passing over the left shoulder of the examiner. After having inspected the thorax by direct illumination, the same procedure should be observed with the patient exposed to oblique illumination. The latter method of examination will often reveal a patch of deficient expansion so slight as to have escaped detection during the examination by direct light. The preferable source of illumination in all cases is daylight, as artificial illumination, aside from altering the appearance of any pigmentation that may be present upon the surface of the thorax, is apt to produce shadows which may result in erroneous conclusions.

THE CHEST WALL

Inspection of the chest wall is apt to reveal changes in the condition of the skin, alterations in the subcutaneous tissues and musculature, changes in the superficial vessels of the thorax, changes in the mammary gland, or abnormalities of the ribs and intercostal spaces.

The *skin* of the thorax in health is smooth and glossy and is lubricated with an adequate amount of sebaceous material. The skin of the adult male thorax is frequently clothed with a variable amount of hair, which is apt to obscure the true state of the skin and which may become a source of error in the application of the various maneuvers which are employed in the physical examination of the thorax.

The *color* of the skin varies normally in different individuals Aside from this normal variation in pigmentation, certain diseases which are not directly referable to the respiratory organs produce more or less characteristic pigmentation of the skin, notably Addison's disease, syphilis, chronic malarial intoxication, hepatic disorders, and following herpes zoster; whereas chlorosis and pernicious anemia lend a greenish or lemon-yellow tint to the skin of the thorax as indeed to that of the entire body. In arthritis of long standing the thoracic integument is shiny and frequently covered with the pustules of acne. In the cachexia of malignant disease it is thinned and presents a yellow, straw-colored tint; while in advanced chronic phthisis the skin of the thorax is dry, with general pallor, and with here and there irregular, yellowish spots of pityriasis versicolor from which it is easy to detach a thin layer of epidermis. The skin of the thorax is subject to the eruptions of the acute, exanthematous fevers; and the skin of this region not infrequently presents scars, the result of trauma or of syphilis.

Occasionally the examiner will encounter silvery striæ, analagous to the striæ developing upon the abdomen during pregnancy in patients who have suffered from thoracic distention during early life and adolescence. The striæ are ordinarily situated upon the posterior and inferior portions of the thorax and pursue a course parallel with the intercostal spaces. They are observed in the train of extensive lobar pneumonia, serofibrinous pleurisy with effusion, chronic ulcerative phthisis, and pneumothorax. In these pathologic states of the thoracic viscera the striæ develop upon the side opposite the disease process which cripples the corresponding lung and leads to vicarious distention of the opposite lung as a consequence of the compensatory emphysema which develops. Gilbert, Troisier, and Menetrier explain their production as the result of rupture of the elastic fibers of the deeper portions of the thoracic integument along the course of the intercostal spaces, a rupture which is especially likely to occur in this class of patients on account of the unusual elasticity and resilience of the thoracic cage at this time of life.

The condition of the *subcutaneous tissues* and *musculature* of the thorax is influenced by the state of general nutrition of the patient, and varies as this is good, moderately good, or poor. The extensive atrophy of these tissues in chronic ulcerative phthisis presents a striking contrast with the prominence of the ribs in this disease, while a similar wasting of these tissues may be the result of chronic pressure from a pleural effusion of prolonged duration.

The *superficial veins* of the thoracic wall, scarcely visible in the normal subject, become engorged and tortuous when intrathoracic lesions interfere with the venous return to the right heart. Thus, engorgement of the superficial veins of the thorax is significant of compression of the large intrathoracic venous trunks by mediastinal tumor, aortic aneurysm, or excessively hypertrophied heart. Similarly, distention of the veins over the lower region of the thorax, communicating with similarly distended veins over th abdomen, and not infrequently associated with the caput medusæ is indicative of stasis in the distribution of the portal vein an inferior vena cava.

The *mammary gland,* an imperfectly developed and rudimentar structure in the male subject, in the presence of pulmonary tuberculosis in this sex has occasionally been observed to undergo a extensive hypertrophy. In other instances hypertrophy of thi gland in the male sex is congenital and devoid of diagnostic significance. In the latter event the glandular hypertrophy is commonl bilateral. In the female subject, on the contrary, hypertrophy o the mammary gland is a normal accompaniment of pregnancy an lactation, and enlargement of the gland also attends malignant disease.

Edema of the thoracic wall may be encountered in a general or local form. General edema of the chest wall attends general an asarca. Moderate edema of the thoracic wall, limited to one side o the thorax, is almost invariably indicative of suppurative diseas within the thoracic cavity. When it develops in the course of serofibrinous pleurisy with effusion, it is suggestive of a purulent alteration of an effusion which has been serofibrinous. When empyem is rapidly extending and rupture through the thoracic wall is imminent, there commonly develops a localized, edematous prominence, usually with moderate discoloration of the integumen *empyema necessitatis.* When this is located upon the left side o the thorax, in close proximity to the heart, the contractions of th heart are apt to provoke systolic pulsation of the contained purulent material, with the consequent production of a condition o *pulsating empyema.*

The *intercostal spaces* are normally slightly depressed, contrasting with the adjacent ribs. In the presence of wasting of th subcutaneous tissues and intercostal musculature as the result o the emaciation of chronic pulmonary tuberculosis, diabetes mellitu or paralysis of the intercostal muscles, the normal depression of th intercostal spaces is accentuated, rendering the ribs unduly prominent. On the other hand, in the presence of extensive pleur effusion and in pneumothorax the intercostal spaces upon the affected side occupy the same plane as their corresponding ribs, i d contrast to the normal depression of these spaces upon th site side of the thorax. Actual bulging of the intercostal spac rarely encountered, save in empyema of extensive duratio *ribs,* not clearly perceptible upon inspection in the norm subject save in the lower axillary and infraaxillary regions, in the presence of chronic wasting disease become conspicuous landmarks upon the surface of the thorax. In rickets, moreover, there is permanent deformity at the junction of the ribs and costal cartilages, constituting the *rachitic rosary* of this disease.

The course pursued by the ribs is suggestive of certain diseases of the respiratory organs. In the alar thorax of chronic ulcerative phthisis the ribs pass sharply downward from the vertebral column, bending sharply upward to meet the sternum, resulting in a very acute subcostal angle. In the barrel chest of hypertrophic emphysema, on the contrary, the ribs pass horizontally forward from the vertebral column with the consequent production of a very obtuse subcostal angle. In rickets, moreover, the ribs are compressed laterally, with consequent diminution in the lateral diameter of the thorax; and in the pigeon breast the ribs are markedly compressed anterior to their angles, resulting in the keel breast of advanced rickets.

The *costal cartilages*, normally elastic and mobile, with advancing age become progressively less resilient through advancing calcification, limiting the freedom of excursion of the thorax during respiration.

THE SIZE AND SHAPE OF THE THORAX

The size and shape of the thorax vary greatly in the normal subject, and are remarkably perverted from the normal state in a number of diseases of the respiratory organs, as well as in certain diseases of other origin, and not infrequently as the result of occupation. Persons who habitually pursue strenuous occupations present a generally overdeveloped thorax, whereas carpenters and cobblers are apt to exhibit the funnel-chest. Similarly, the thorax of the person engaged in clerical work is apt to present an elevation of the right shoulder, which is quite normal in this instance. Moreover, in certain diseases, notably in rickets, hypertrophic emphysema, and chronic ulcerative phthisis, the thorax becomes permanently fixed in deformity, and a diagnosis is frequently suggested during a casual inspection by the characteristic configuration of the thorax.

THE NORMAL THORAX

The various modifications to which the normal thorax is subject can only be recognized after extensive clinical experience. Essentially, the size and shape of the thorax are governed by the degree of intrathoracic contents, and in the normal subject the thorax presents quite a varied picture in different cases.

Two general types of normal thorax may be distinguished; namely, the *inspiratory type* of thorax of the physically robust subject; and the *expiratory type* of thorax of the subject of moderate physical development. In the latter the thorax is rather shallow with moderate anteroposterior flattening in the infraclavicular and mammary regions, slightly elongated, and presents a rather acute subcostal angle; whereas in the former the chest is deep, the anteroposterior diameter of the thoracic cavity approaching but not attaining to the dimensions of the lateral diameter, and with quit an obtuse subcostal angle.

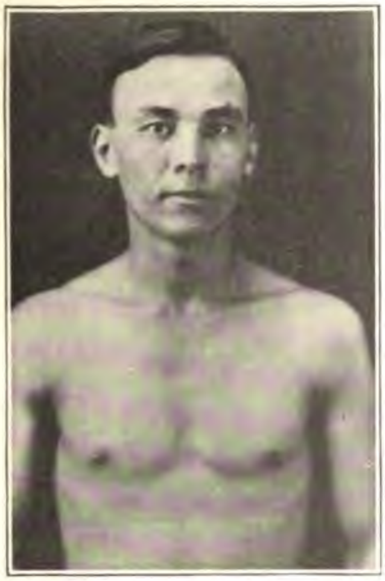

Fig. 13.—Normal thorax (in repose).

In the normal subject the shoulders usually occupy the same level though moderate dropping of one shoulder is not infrequent. Th clavicles are not unduly prominent; but moderate depression of th supraclavicular and infraclavicular fossæ is not incompatibl with perfect physical well being. The two halves of the thora are seldom perfectly symmetrical, as the right side is usually mor fully developed than is the left side owing to the greater employment of the right arm in right-handed individuals.

obstruction of the upper respiratory passages by hypertrophied tonsils or adenoid vegetations, in children; while deviations from the normal contour of the thorax in the adult, on the contrary, are caused by, and are secondary to, pathologic changes in the thoracic viscera, such as collapse or fibroid retraction of a lung, excessive inflation of the lungs by hypertrophic emphysema, the presence of aneurysm of the thoracic aorta, excessive cardiac hypertrophy, or the development of an intrathoracic neoplasm. From these considerations, it follows that deformity of the thorax may be bilateral, unilateral, or local, depending upon the nature of the underlying etiologic factors.

Bilateral Deformities

The Emphysematous Thorax (Barrel-chest).—In hypertrophic emphysema the thorax presents characteristic deviations from the normal contour, constituting the barrel-chest of this disease. The thorax is increased in all of its diameters, but particularly in the anteroposterior diameter, which in marked contrast to the normal thorax, exceeds the transverse diameter. The thorax is thick and relatively short, and is maintained in a position of distention which exceeds that which normally obtains at the completion of full inspiration. The thorax upon cross section is almost circular, with the maximum degree of enlargement corresponding to the level of the central point of the sternum.

The thorax presents the greatest degree of distention in the upper and central portions, for the reason that the chronic catarrhal inflammation of the smaller bronchioles throughout the voluminous bases presents an obstacle to the free ingress of the inspired air to these portions of the lungs, whereas the apices and anterior borders of the lungs expand compensatorily. For the same reason, the excursion of the diaphragm during respiration is limited, throwing an increased burden upon the upper intercostal muscles and the accessory muscles of respiration, all of which tends to accentuate the discrepancy between the expansion of the upper and lower halves of the thorax. However, owing to the acquired rigidity of the costal cartilages in the emphysematous thorax, there is little expansion of the chest; but in compensation for this deficiency on the part of the normally resilient costal cartilages, the thorax during respiration rises and falls *en masse*.

The ribs in the upper half of the thorax pass almost horizontally forward from the vertebral column to meet the sternum, while the normal obliquity of the lower ribs is likewise diminished, resulting in a very obtuse costal angle, and a corresponding increase in the outlet of the thorax. Owing to the horizontal course of the ribs and the general distention of the thorax, the intercostal spaces are abnormally wide, and peculiarly hard and unyielding upon palpation. Not infrequently there is visible retraction of the lower intercostal spaces during inspiration.

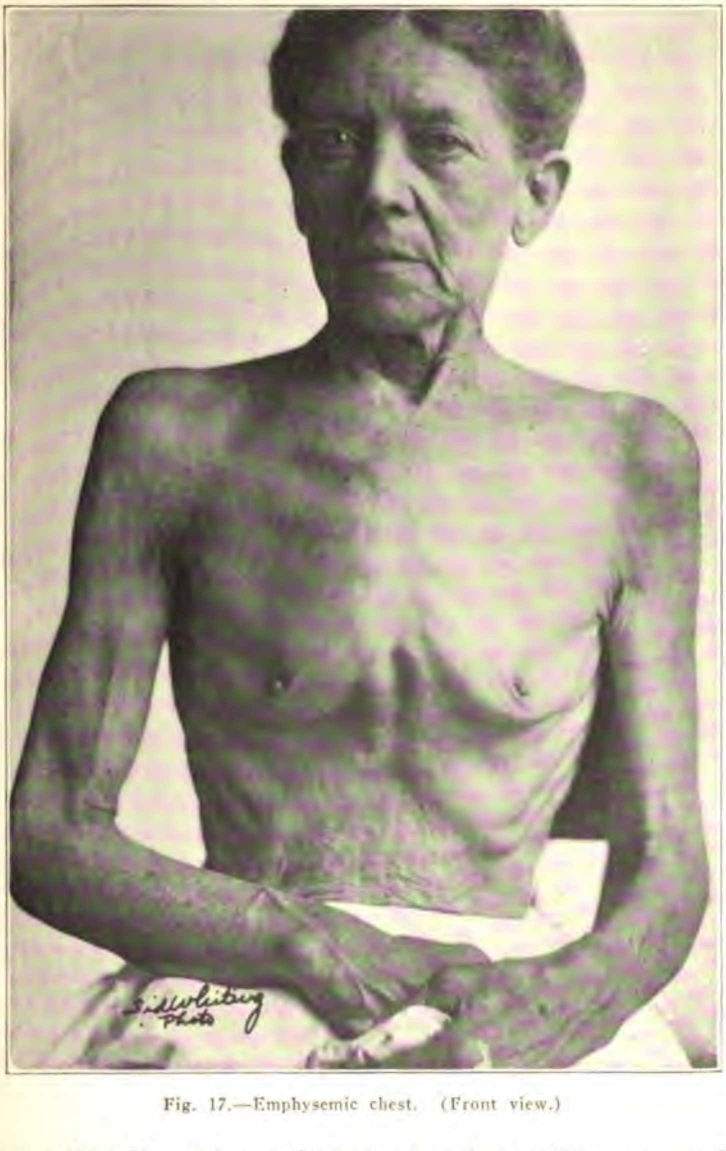

Fig. 17.—Emphysemic chest. (Front view.)

As a rule, the sternum occupies the same plane as the costa cartilages, without noticeable bulging or recession, though th bone is not infrequently bent slightly at the junction of the manubrium and gladiolus, resulting in an unduly prominent angulus Ludovici.

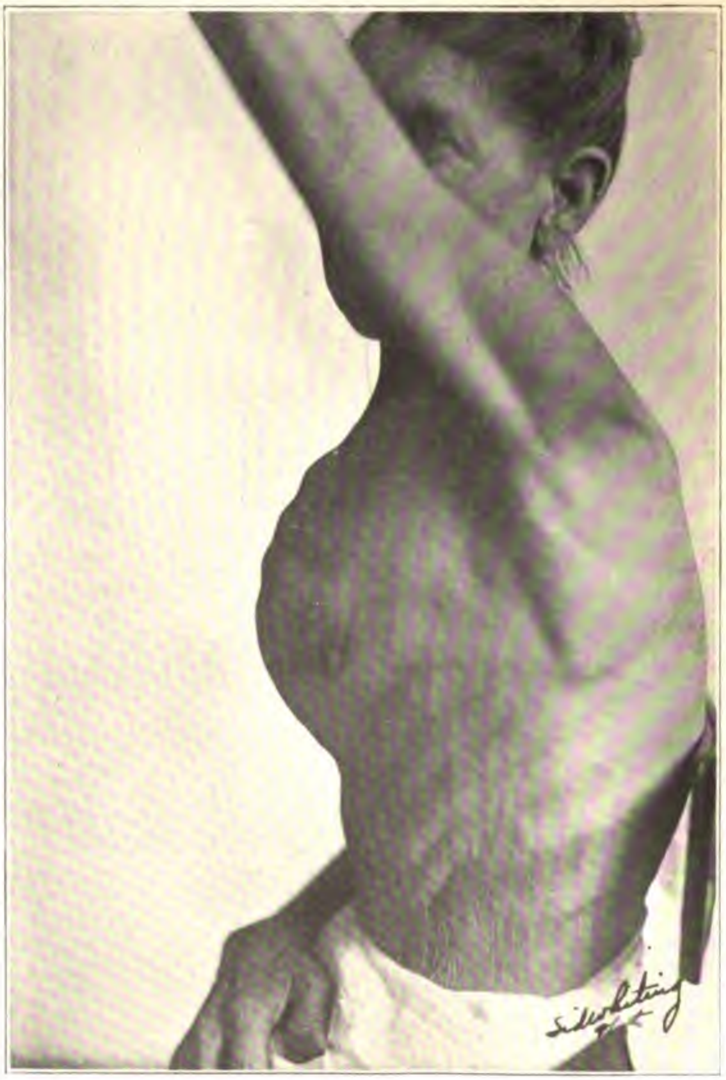

Fig. 18.—Emphysemic chest. (Lateral view.)

The scapulæ are closely applied to the back of the bony thorax The normal dorsal curve of the vertebral column is accentuated

simulating kyphosis in the extreme case. The clavicles are el
vated, and the neck is short and thick, often with conspicuo
engorgement of the cervical veins. The sternomastoids are u
duly prominent and the head is slightly thrown backward in tl
effort to bring the accessory muscles of respiration into play.

The condition of the supraclavicular and infraclavicular foss
is variable. Frequently these spaces have disappeared and tl
integument occupies the level of the clavicle or even presents
local bulging.

Kyphosis acquired during laborious occupations or developir
as the result of dorsal caries may be mistaken for the barrel-che
of emphysema, as may also thoracic distention due to maligna
disease of the thoracic viscera, or thoracic deformity due to e
tensive pleural effusion or to enlargement of the liver or of tl
spleen.

The Phthisical Thorax (Alar, Pterygoid, or Paralytic Thorax
—The phthisical thorax presents a marked contrast to the er
physematous chest. In the phthisical thorax the anteroposteri

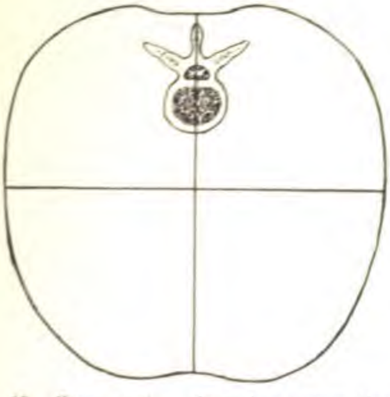

Fig. 19.—Cross section of emphysematous thorax.

diameter is greatly diminished, and the thorax is long and fla
presenting a picture of extreme emaciation, owing largely t
wasting of the pectoral and deltoid muscles. The clavicles stan
out prominently, in striking contrast to the deep recession of tl
supraclavicular and infraclavicular fossæ above and below tl
bones.

The inner borders of the scapulæ stand out upon the posteri
acic wall like wings, hence the name "alar thorax" which
ently applied to this type of thoracic deformity. The win
scapulæ enjoy an abnormal mobility upon the posteri

thoracic wall, owing to atrophy of the investing musculature. The shoulders, which seldom occupy the same level, are inclined forward, while the head habitually droops anteriorly.

The ribs pursue a very oblique course downward from the vertebral column and bend sharply upward in front of their angles to meet the sternum, producing a very acute subcostal angle. The intercostal spaces are wide; and the intercostal muscles are wasted, yielding readily to palpation with the finger-tips. The elongated, flat thorax is surmounted by a long, tapering neck in which the sternomastoid muscles and the larynx stand out prominently.

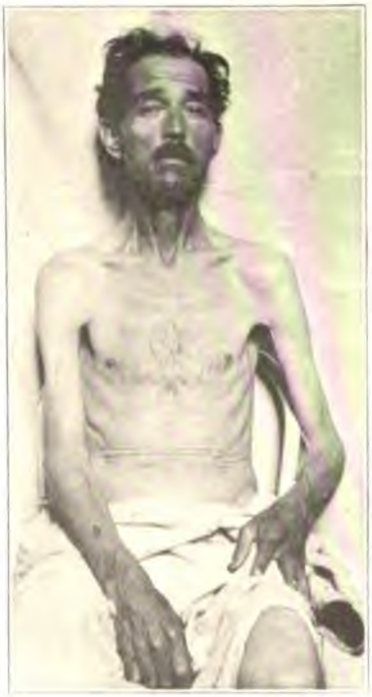

Fig. 20.—Phthisical thorax.

The phthisical thorax in its full development is one of the signs of chronic ulcerative phthisis. In its partially evolved form it is encountered in inanition and in subjects with a predisposition to phthisis.

The Rachitic Thorax.—Advanced rickets is accompanied by a characteristic deformity of the thorax. In this type of thoracic deformity the anteroposterior diameter is increased, while th lateral diameter is diminished as the result of muscular actio upon the abnormally yielding ribs, causing the sternum to ju forward and to assume an unduly prominent position. A cros

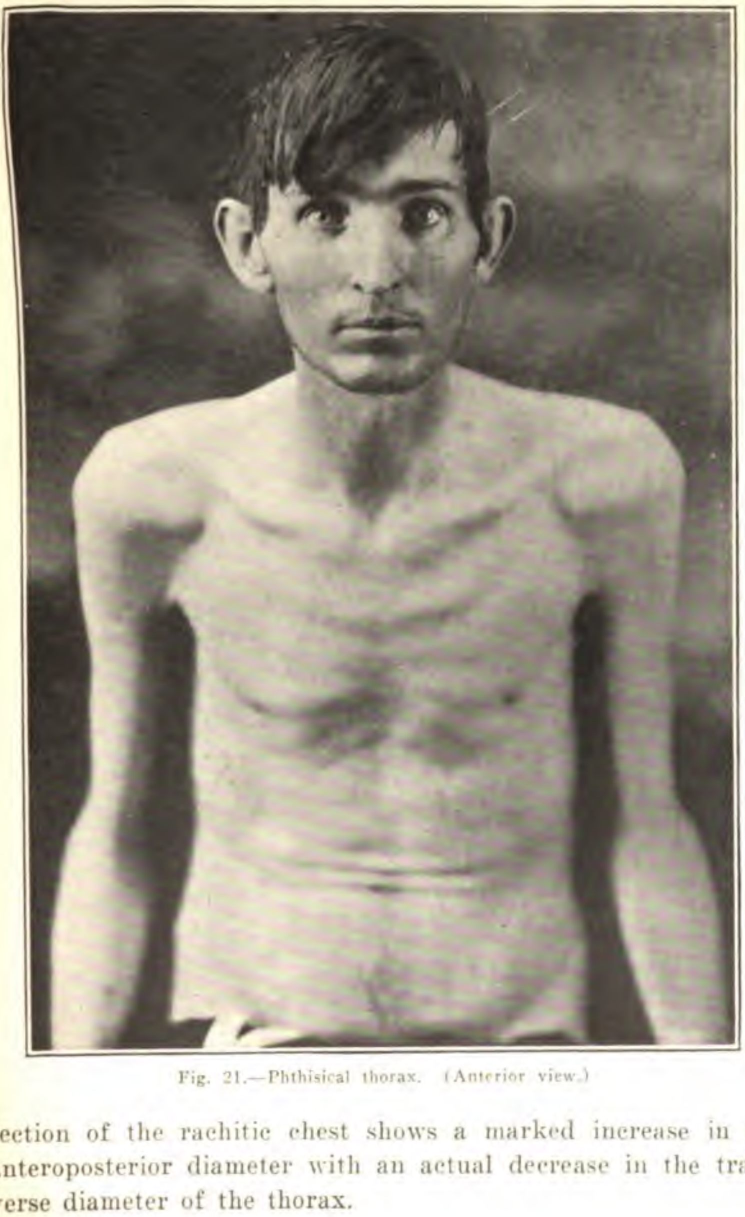

Fig. 21.—Phthisical thorax. (Anterior view.)

section of the rachitic chest shows a marked increase in th anteroposterior diameter with an actual decrease in the tran verse diameter of the thorax.

At the junction of the ribs with their costal cartilages t rachitic thorax presents a series of nodular swellings, due to e largement of the osteocartilaginous junctional tissues, the *rachi rosary.* As a result of the lateral compression of the thorax, t

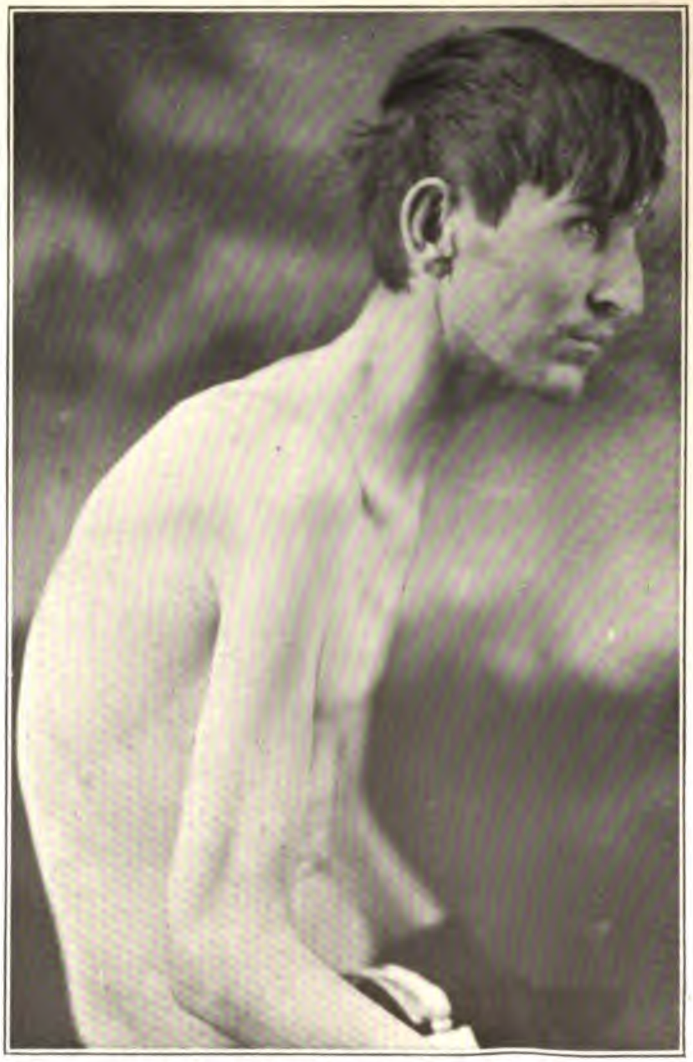

Fig. 22.—Phthisical thorax. (Lateral view.)

costal angle is abnormally acute. Not uncommonly the low ribs flare outward anteriorly. The rachitic thorax is attend by various types of spinal curvature, as kyphosis, lordosis, scoliosis.

The rachitic chest is not significant of any disease of the respiratory organs; but the compressed thorax is not sufficientl capacious for the lungs to properly expand and to attain thei full development, and hence this type of thoracic deformity predisposes to disease of the organs of respiration.

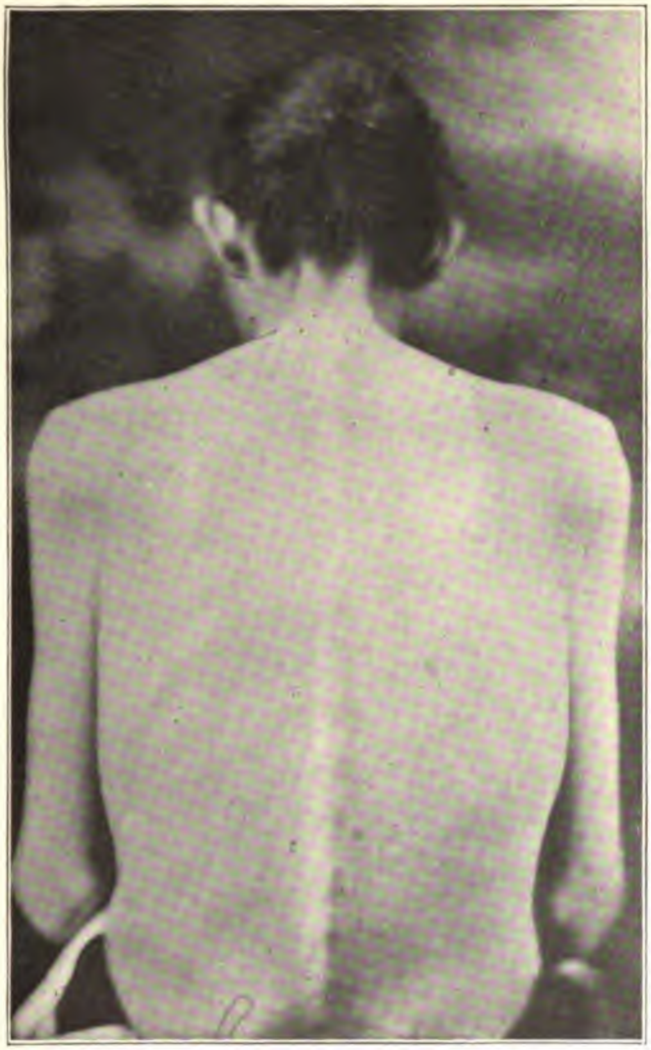

Fig. 23.—Phthisical thorax. (Posterior view.)

Harrison's Sulcus.—Harrison's sulcus is a groove or depressio extending downward and outward upon either side of the thora from the ensiform cartilage toward the infraaxillary regions. It is often a sign of early rickets; it almost invariably accompanies the rachitic thorax; and it is also caused in early life by obstructive lesions of the upper air passages, in which event it is to be attributed to the external atmospheric pressure compressing the soft ribs which are not supported by full inflation of the lungs. The deformity is particularly common in the negro race.

The Pigeon-Breast. (Keel-Breast: Pectus Carinatum).—In this type of thoracic deformity the ribs are compressed and straightened in front of their angles, causing the sternum to jut forward and to become unduly prominent. Upon cross section the thorax is roughly triangular. The anterior portion of the thoracic cavity is encroached upon by the incurvation of the ribs, while the posterior portion of the cavity is compensatorily voluminous. Harrison's sulcus is frequently present upon the lateral regions of the thorax.

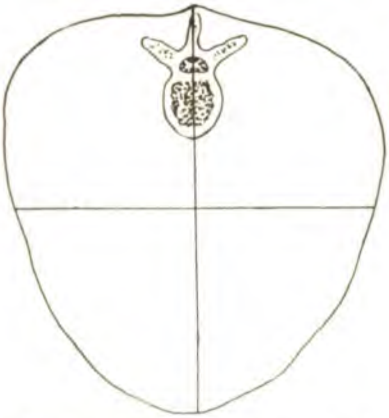

Fig. 24.—Cross section of rachitic thorax.

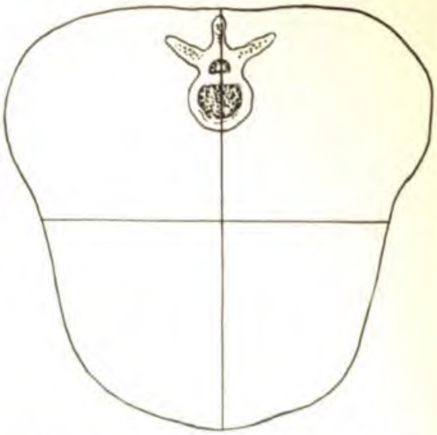

Fig. 25.—Cross section of pigeon breast.

The pigeon-breast occurs most frequently in cases of advanced rickets; but it may also be produced by the paroxysms of pertussis or by greatly hypertrophied tonsils during early life.

The Funnel-Chest.—In this type of thoracic deformity the lower end of the sternum is depressed, the hollow or depression occasionally extending as high as the third rib. The funnel-chest is in the vast majority of instances a congenital deformity; it has been noted in connection with advanced rickets; and it occasionally develops as the result of an occupation which requires the application of an instrument constantly against the lower portion of the sternum as in the case of the cobbler or the ter. The funnel-chest is not essentially a sign of isease; but, by decreasing the capacity of the thorax, es to disease of the bronchopulmonary system.

rmity of the Spine.—Kyphosis, lordosis, and scolio as part and parcel of the thoracic deformities att

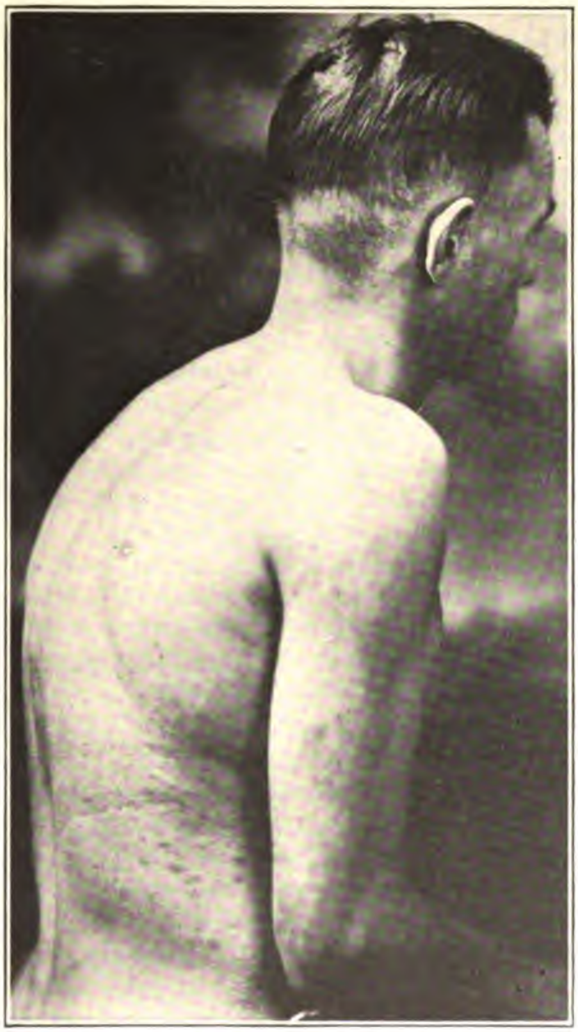

Fig. 26.—Kyphosis due to vertebral caries.

s. Lateral deviation of the spine is noted in the p e pleural effusions, extensive empyema, and in the p of intrathoracic neoplasm. In vertebral caries kyphosis is a frequent finding.

Unilateral Deformities

Unilateral Enlargement.—Enlargement or bulging of one side of the thorax is caused by an increase in the size of the visceral content of the corresponding side, or by the presence of abnormal material, which may be air, gas, serum, pus, or blood. The enlargement may be significant of compensatory emphysema of one entire lung, arising as the result of vicarious distention of the lung to compensate for crippling of the opposite lung by cavitation, compression, or fibrosis, in which event the retraction of the diseased side of the thorax adds materially to the apparent bulging of the emphysematous side. The presence of air or gas in the pleural cavity in open or closed pneumothorax produces unilateral bulging of varying degree. The compression of the lung in these cases also results in compensatory emphysema of the opposite lung; but the latter does not attain such a degree as to equalize the enlargement of the two sides of the thorax. Extensive pleural effusion, hemothorax, empyema, and hydrothorax cause similar disproportion between the two halves of the thorax; and in these cases, in which the enlargement is due to the presence of fluid, the bulging is most pronounced in the lower portion of the thorax; the costal angle is rendered more obtuse; and the intercostal spaces occupy an equal plane with the ribs or show slight bulging.

In unilateral enlargement of the thorax the circumference and anteroposterior diameter of the affected side are increased, and the intercostal spaces are wider than normal. The shoulder upon the affected side is slightly elevated, and the vertebral column deviates toward the side of the bulging.

Unilateral Diminution.—Diminution of the size of one-half of the thorax is indicative of a corresponding diminution in the size of the intrathoracic contents upon the affected side. Chronic pulmonary tuberculosis of the corresponding lung is the most prolific cause of this deformity. In other instances it is due to bronchial obstruction with subsequent collapse of an extensive portion of the lung. Fibrosis of the lung in the course of chronic interstitial pneumonia, fibroid phthisis, and pulmonary syphilis results in unilateral retraction or flattening of one side of the thorax. A further causative factor in the production of this type of thoracic deformity is the traction of adhesions which bind the visceral to the parietal pleura, or total obliteration of tl potential pleural cavity, the result of chronic adhesive pleuris

All of these conditions are attended by compensatory emphysen of the opposite lung, thus accentuating the disproportion betwee the two sides of the thorax. In this deformity the circumferen of the affected side is diminished, as is the anteroposterior d ameter; whereas the transverse diameter is increased. In cas due to chronic pulmonary tuberculosis, the retraction is mo marked upon the upper half of the thorax; whereas in cases d pendent upon chronic adhesive pleurisy the deformity is mo noticeable over the lower thoracic region.

In addition to the diminution in the size of the affected sid the corresponding shoulder droops, and the vertebral colum deviates, with its concavity directed toward the retracted sid This curvature of the spine is apt to prove a source of error the casual examination of cases presenting minor degrees of ur lateral retraction, as lateral spinal curvature from other caus may simulate unilateral diminution of the thorax in its absenc The intercostal spaces upon the retracted side of the thorax a narrowed; and, in cases of extreme retraction, the ribs may act ally overlap in the lower thoracic region.

Local Deformities

Local deformity of the thorax may have reference to chang in the organs of respiration or to alterations in the circulato system. Those which are referable to the former possess a va able significance, depending upon the location upon the thorac surface in which they occur and upon their general physical cha acteristics.

Local Prominence.—Upon the anterior or lateral thoracic wa a local prominence is indicative of a localized serofibrinous pleu isy; a large neoplasm of the mediastinal structures, lung, or che wall; or of a large pulmonary cavity which is filled with flui A local prominence which is associated with edema or discolor tion of the integument of the thorax signifies empyema nece sitatis. Rarely congenital diaphragmatic hernia is responsib for local prominence of the chest wall. Undue prominence of tl sternum, when not caused by aneurysm of the aortic arch, is fr quently due to malignant disease of the mediastinal glands. Loc prominence of the left hypochondriac region points to splen enlargement, while a similar enlargement over the right hyp chondriac region is indicative of hepatic enlargement or of a low right-sided pleural effusion.

Upon the posterior thorax a local prominence in the median line, overlying the vertebral column, accompanies vertebral caries and spina bifida; while upon either side of the median line in the paralytic thorax the inner borders of the alar scapulæ stand out prominently.

Local Retraction.—Undue depression of the supraclavicular and infraclavicular regions, with unduly prominent clavicles, points in the first instance to apical pulmonary tuberculosis, and secondarily to fibrosis of the lung or to traction by pleural adhesions. Local retractions upon the anterior thoracic wall, adjacent to the lateral sternal borders, frequently indicate pulmonary excavation due to chronic ulcerative phthisis or to bronchiectasis; while a circumscribed area of flattening or of moderate depression upon the posterior wall of the thorax accompanies pulmonary abscess. Local retraction over the lower portion of the sternum constitutes the essential feature of the funnel-chest, whereas upon the lateral walls of the thorax Harrison's sulcus is frequently noted in rachitic patients. As a general rule, local retractions in the lower anterior and lateral thoracic regions are the result of traction by pleural adhesions.

Movements of the Thorax

A study of the movements of the thorax in health and in disease constitutes an important part of every physical examination. These movements are influenced by the rate, rhythm, and the type of respiration obtaining in a given case; and the movements are influenced and modified by disease arising within the thorax or in other portions of the body. Observation of the thoracic mobility reveals the degree of expansion of the thorax, as well as the diaphragmatic phenomenon of Litten. Finally, circumscribed mobility of the thorax is noted in the form of normal and abnormal pulsations.

Respiratory Movements of the Thorax.—The respiratory movements of the thorax comprise an inspiratory excursion and an expiratory recession, the latter being followed by a slight pause during which the thorax is in a state of repose. The inspiratory movement is an active process, which is initiated and executed by muscular contraction; whereas the expiratory recession is a passive movement, the thoracic walls receding upon the cessation of the muscular action. Of the two phases, the expiratory rece sion is of longer duration than is the inspiratory excursion.

The *frequency* of the respiratory movements of the thorax health varies with the age and sex of the subject. In the norm adult the frequency is fourteen to eighteen respirations p minute. In the newly born, on the contrary, the respiratory ra

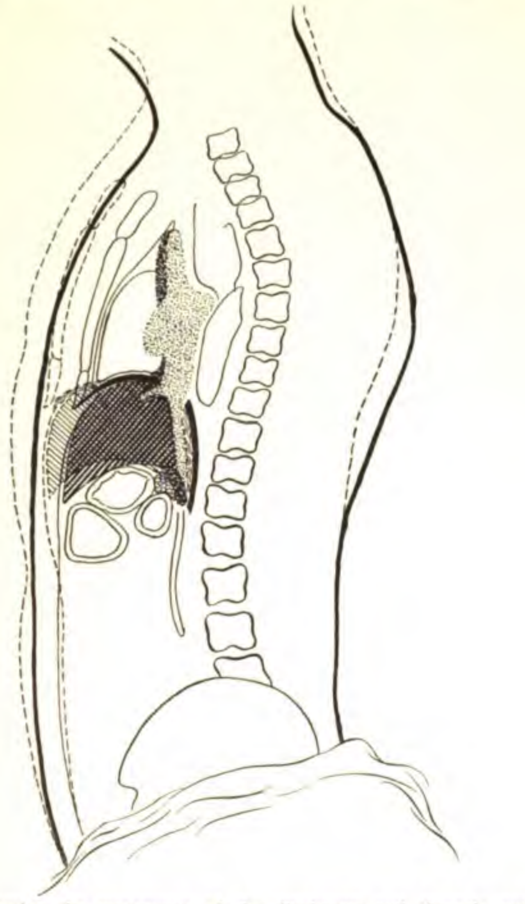

Fig. 27.—Illustrating the movements of the diaphragm and thoracic and abdomi walls, as well as the change in position of the intrathoracic and intraabdominal visce during respiration of the abdominal type. The movements are from the solid lines expiration to the broken lines on inspiration. (Pottenger, after Hasse.)

is approximately forty-four to the minute, while at five years age it averages twenty-six respirations per minute. The respir tory rate in the female subject is normally slightly in excess that which obtains in the male.

The *character* of the respiratory movements of the thorax al [illegible] subject, and two types of normal [illegible] namely, the *costal type*, and the *costo-* [illegible] respiration.

[illegible] subject and during childhood the costal type [illegible] in which the thoracic movement is much [illegible] that of the abdomen. In this type of res-

[illegible] the diaphragm and thoracic and abdominal walls, [illegible] intrathoracic and intraabdominal viscera, when [illegible] are pronounced. The movements are from [illegible] lines of inspiration. (Pottenger, after Hasse.)

[illegible] portion of the thorax, above the level of the [illegible] maximum degree of expansion, the lower thor- [illegible] conspicuously; hence the name "upper [illegible] is sometimes employed to describe it. The [illegible] is but a minor degree in this movement so that there is little or no noticeable epigastric bulging during inspiration.

In the adult male subject, on the contrary, the respiration is of the costoabdominal type, in which the maximum expansion is noted over the lower thorax and the upper abdomen. The diaphragm is a prime factor in the execution of this type of respiration, and by virtue of its descent during inspiration, this phase of the respiratory cycle is marked by excursion of the lower thoracic walls and epigastric bulging, while the costal angle becomes more obtuse. At the same time the sternum is elevated; the ribs assume a more horizontal position; and there is an increase in both the anteroposterior and the transverse diameters of the thorax.

Yet while these distinct types of normal respiration obtain in the two sexes, an exaggeration of either the costal or the costoabdominal type, or the substitution of the one for the other, is abnormal; and such alteration possesses definite diagnostic significance. Thus, exaggeration of the costal type of respiration in the female subject or the presence of this type in the male is indicative of immobilization of the diaphragm by subphrenic pressure or of paralysis of this important muscle. Excessive subphrenic pressure, preventing the descent of the muscle during inspiration may be due to ascites, large abdominal tumor, tympanites, or peritonitis.

Similarly, accentuation of the costoabdominal type of respiration in the male or its presence in the female subject points to premature calcification of the costal cartilages or scleroderma, destroying the normal elasticity of the upper thorax; or it may signify inhibition of thoracic excursion due to the pain of an acutely inflamed pleura, of pleurodynia, or of a recent fracture of a rib. Such a reversal of the normal type of respiration is also noted in the presence of extensive disease of the thoracic viscera, notably in massive pneumonia, bilateral pleurisy with effusion, pulmonary tuberculosis, or pulmonary neoplasm.

The Diaphragmatic Phenomenon (Litten's Sign).—At the completion of expiration the upper surface of the diaphragm is in apposition with the lateral walls of the thoracic cavity from the seventh to the tenth ribs. During inspiration the diaphragm, which at the completion of expiration is in close apposition with the chest wall in its lower portion, becomes separated from the thoracic parietes gradually and progressively in its descent. The gradual separation of the two apposed surfaces during inspiration causes a slight undulation or "shadow" to pass downward upon the lateral thoracic walls from the seventh to the tenth ribs when the patient is exposed to oblique illumination. During expiration, as the diaphragm in its ascent again adapts itself to the chest wall, there is an ascending undulation in the same area, which, however, is not as readily perceptible as is the descending "shadow" which occurs during inspiration.

To elicit Litten's sign the patient is placed in the dorsal posture with the head comfortably elevated, and with the hands clasped above the head, with the feet directed toward the source of illumination, preferably an open window, the light from which should fall obliquely upon the side under observation. The examiner assumes a position near the feet of the patient, with his back toward the source of illumination; whereupon in a normal thorax the undulation is perceived to descend for the space of two inches or more during inspiration and, under favorable circumstances, to ascend to an equal degree during expiration.

Restriction of, or abolition of the "shadow" points to an abnormal condition within the thorax or abdomen which interferes with the free excursion of the diaphragm or to paralysis of this muscle. In the thorax it may signify pneumothorax, pneumonia, pleurisy with effusion, adhesions between the visceral and parietal pleura, hypertrophic emphysema, intrathoracic neoplasm or pulmonary tuberculosis, interfering with the proper inflation of the lungs. In the abdomen abolition of the undulation may be indicative of increased subphrenic pressure from hepatic or splenic enlargement, subphrenic abscess, ascites, or large abdominal tumor.

Pathologic Respiratory Variations.—In the presence of disease of the thoracic viscera and in many instances of disease of distant portions of the bodily economy, as well as in numerous constitutional affections, the respiration presents typical variations from the normal rhythm and type. The predominant change may be an increase or a diminution in the frequency or depth of respiration, or it may consist in a prolongation of one or the other phase of the respiratory cycle. In other instances the respiration becomes stridulous, sterterous, or it assumes the meningeal or Cheyne-Stokes type. In yet other instances the respiration becomes so labored as to be distinguished as dyspnea or orthopnea, which may or may not be attended by cyanosis.

Rapid Respiration (Polypnea). Simple increase in the rate of the respiratory movements of the thorax is observed as a result of active physical effort, during mental and emotional excitement, and not infrequently is induced by the consciousness on the part of the patient of the examination, without possessing untoward significance. Febrile movement is attended by polypnea, as ar certain forms of cerebral disease and hysteria. Pulmonary lesion which decrease the air space of the lungs as do the consolidation of pneumonia and tuberculosis; compression of the lung b pneumothorax, pleurisy with effusion, or mediastinal tumor; an elevation of the diaphragm by subphrenic abscess, ascites or abdominal neoplasm; all result in acceleration of respiration. During imperfectly compensated valvular heart disease the respirations are rapid; and during the course of diabetes mellitus an uremia elevation of the respiratory rate of varying degree i noted.

Slow Respiration (Oligopnea).—Diminution of the respirator movements of the thorax, the number falling below fourtee respirations in the minute, is observed in states of collapse an in the coma of uremia or diabetes. Increased intracranial pressure from tumor, abscess, hemorrhage, or meningitis is attende by an abnormally slow respiratory rate. The respiratory movements are also diminished during the course of infectious disease which are associated with mental torpor. In these states, in addition to the diminution in the frequency of the respiratory movements, the rhythm of the respiration is usually disturbed.

Prolonged Inspiration.—Inspiration is unduly prolonged i tracheal and laryngeal obstruction from spasm, compression new growth, or foreign body. In these states the lower intercostal spaces and the upper abdomen are retracted during th violent inspiratory effort.

Prolonged Expiration.—The expiratory phase of the respiratory cycle is unduly prolonged in hypertrophic emphysema and i bronchial asthma, the muscles of expiration being called int play in the effort to expel the tidal air from the lung, substitutin an active for a passive process. In the paroxysm of bronchia asthma, in addition to the prolongation, expiration is dotted wit numerous râles.

Stertorous Respiration (Snoring Breathing).—During the com of uremia, diabetes, and apoplexy respiration is frequently attended by stertor. The same type of respiration is frequentl noted in cases of palatal paralysis. Less frequently stertorou respiration is encountered in the presence of large postpharyngea abscess, with extensive adenoid vegetations in the nasopharynx in chronic tonsillar hypertrophy, and in quinsy. Aside fro these local causes, stertorous respiration is frequent in the com the patient is exposed to oblique illumination. During expiration, as the diaphragm in its ascent again adapts itself to the chest wall, there is an ascending undulation in the same area, which, however, is not as readily perceptible as is the descending "shadow" which occurs during inspiration.

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resulting from poisoning from illuminating gas, alcohol, opium, or other narcotic drug.

Stridulous Respiration (Hissing Breathing).—Stridulous respiration is indicative of laryngeal or tracheal obstruction by infiltration, compression, new growth, or foreign body. It is produced by compression of these structures by enlarged glands, mediastinal tumor or an excessively hypertrophied heart. Hissing breathing also occurs in spasm and edema of the glottis, which is apt to develop in the course of syphilis or tuberculosis of the larynx, during diphtheria and acute laryngitis, or as a complication of one of the acute infectious fevers. Stridor also accompanies the paroxysms of pertussis and the attacks of laryngismus stridulus, the stridor in these instances being more pronounced during, or entirely limited to, inspiration. The stridor of laryngeal obstruction is as a rule accompanied by aphonia.

Cheyne-Stokes Respiration.—In this type of respiration, following a period of transient apnea, the respirations become progressively deeper until a maximum depth is attained, whereupon they gradually become more shallow to finally terminate in another apneic period. The period of apnea lasts from ten to twelve seconds as a rule, and during this time the patient is apt to become unconscious, with cyanosis, slowing of the pulse rate, and myosis. Persisting for a period of time varying from a few hours to several months, this type of respiration is of grave prognostic significance. It is frequently a sign of impending dissolution. Associated particularly with cerebrospinal meningitis, cerebral tumor, and apoplexy, Cheyne-Stokes respiration is also frequently noted during the coma of uremia and diabetes, and more rarely with arteriosclerosis and chronic valvular heart disease, and very rarely indeed it develops during the course of typhoid fever and lobar pneumonia.

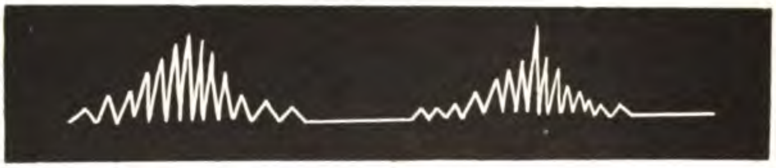


Fig. 29.—Cheyne-Stokes respiration.

Biot's Respiration (Meningeal Breathing).—In this type of pathologic respiration there are periodic interruptions in the respiratory sequence, during which the patient is apneic. The periods of apnea vary in duration and recur at irregular intervals. The periods of apnea lack the definite regularity which is noted in Cheyne-Stokes respiration. In addition, there is marked irregularity in the depth and rhythm of the individual respirations.

Occurring most frequently during the course of meningeal inflammation, Biot's respiration is also observed occasionally in cerebral hemorrhage and tumor, and during the periods of somnolence of acute infectious fevers.

Dyspnea

Dyspnea, difficult or labored breathing, is recognized clinically by the increased frequency of the thoracic excursions and by the participation of the accessory muscles of respiration in the movements. Dyspnea may be present in varying grades, ranging from a slight increase in the frequency of the respirations to extreme difficulty accompanied by blueness of the lips and finger-tips (cyanosis).

Dyspnea may be entirely limited to inspiration, as when the air passages are obstructed by a foreign body, or it may be expiratory as is the case in hypertrophic emphysema and bronchial asthma. Usually there is present a combination of inspiratory and expiratory dyspnea.

Dyspnea may arise as a result of deficient aeration of the blood content of the lungs arising as a result of an obstruction to the free ingress of air to the lungs, the causes of which are indeed varied. The obstruction may reside in hypertrophy of the turbinates or in adenoid vegetations in the nasopharynx, retropharyngeal abscess, or excessive tonsillar hypertrophy. The obstruction may reside in the larynx in the form of edema of the glottis, spasm of the rima glottidis, cicatricial stenosis from syphilitic laryngitis, or from lodgment of a foreign body in this portion of the air passages. Inspiratory dyspnea may also be caused by partial stenosis of the trachea, occurring as a sequence of cicatricial stenosis of the tube of intratracheal origin, or from compression of the trachea from without by an enlarged thyroid gland, a mediastinal tumor, or aneurysm of the aorta. Similarly, inspiratory dyspnea may arise from obstruction of a principal bronchus by cicatrix, pleural adhesions, or foreign bodies.

In all of these states the interference with respiration is inspiratory, as recognized by the prolongation of the inspiratory phase of the respiratory cycle. Inspiration is prolonged unduly frequently with the development of an audible stridor, whereas expiration is brief and is attended by little movement of the thoracic walls.

Dyspnea involving principally expiration is noted in subjects of hypertrophic emphysema and during the paroxysm of bronchial asthma. In these diseases following upon a fairly rapid inspiration, there succeeds an unduly prolonged expiratory phase during which the inspired air is ejected tardily and with obvious effort upon the part of the subject of the disease. In bronchial asthma the expiratory phase is dotted with numerous piping sibilant and snoring, sonorous râles.

When the pulmonary alveoli are filled with the consolidations of pneumonia or phthisis, and when the lung is compressed by a large pleural effusion or mediastinal tumor, dyspnea develops as a result of an actual diminution of the aerating surface of the lung in which the circulating blood is exposed to the action of the aerial content of the lungs. A similar state obtains in the presence of extensive effusion into the peritoneum or in the presence of a large intraabdominal tumor or marked tympanites, conditions in which the diaphragm is displaced upward, partially immobilizing the thorax.

Dyspnea also develops in the presence of extreme weakness of the muscles concerned in the respiratory excursion of the thorax or with paresis of these muscles. A somewhat analogous state obtains when the thoracic excursions are voluntarily inhibited as a result of the pain incident to an acute fibrinous pleurisy or a fractured rib.

One of the most productive causes of dyspnea is to be found in valvular heart disease, in which regurgitant lesions of the left heart produce passive congestion of the pulmonary circulation. A similar state is induced by mechanical congestion of the lungs, in which the pressure of new growths or enlarged glands upon the pulmonary veins retards the flow of blood from the lungs to the heart. Under these circumstances dyspnea is extreme, amounting to orthopnea.

Acute infectious fevers associated with toxemia are productive of dyspnea, arising from the action of circulating toxins upon the respiratory centers. A more serious dyspneic state follows upon the action upon these centers of the toxins of uremia and diabetes, as evinced by the excessive dyspnea designated as the "air hunger" of Kussmaul.

Primary or secondary anemia, associated with a marked diminution in the hemoglobin or oxygen-carrying content of the blood occasions dyspnea, which may amount in certain cases to *orthopnea,* an extreme grade of dyspnea in which the subject is able to breathe only in the erect or sitting posture.

Cyanosis.—The conditions which result in cyanosis are so closely related to the causes of dyspnea, and the two conditions are so frequently concomitant signs of disease of the thoracic viscera that the significance of cyanosis may most logically be discussed in this connection. The dusky, bluish hue of the integument in this condition is due to deficient oxygenation of the blood, to which is frequently added a variable degree of venous stasis.

The causes of cyanosis which are referable to the respiratory organs may have their site at any point from the upper air passages to the remotest ramifications of the bronchioles. Any interference with the free ingress of air to the pulmonary parenchyma, such as laryngeal or tracheal stenosis from infiltration, pseudomembrane, new growth, compression, or foreign body results in deficient aeration of the blood content of the lung and consequent cyanosis. Conditions which limit the air space of the lungs by accumulation within the infundibula or compression of the lung from without, result in cyanosis. Cyanosis of varying degree attends extensive consolidations of pneumonia and tuberculosis in the late stages, as well as edema of the lungs; whereas, a massive pleural effusion, hemothorax, pneumothorax, or large mediastinal tumor, by compression and immobilization of the lung leads to a similar derangement.

The principal extrapulmonary cause of cyanosis is to be found in valvular heart disease, congenital or acquired, with imperfect compensation, particularly when associated with loss of vasomotor tone. However, compression of the pulmonary veins by large mediastinal tumor, aneurysm of the thoracic aorta, or extensive pericardial effusion, acting in a similar manner, results in cyanosis of varying degree. Moreover, the ingestion of excessive doses of coal tar products produces cyanosis which may be mistaken for cyanosis of pulmonary or cardiac origin.

Cyanosis may be manifested in varying grades, and it may be general or local in its manifestations. General cyanosis is present only in the presence of grave respiratory or circulatory disease, or after overdosage with coal tar derivatives. Less extreme des of cyanosis become manifest as local cyanosis, which is pally noticeable in the lips, cheeks, buccal mucous membrane, ears, and beneath the finger-nails. The surface temperature of cyanotic areas is invariably reduced.

Abnormalities of Thoracic Expansion

Abnormalities in the degree of expansion of the thorax may be manifest in the form of bilateral or unilateral variations from the normal, or the anomaly may be circumscribed to certain, definite areas of the surface of the thorax. The normal degree of thoracic expansion is a variably quantity, varying with the degree of physical development of the individual and with the vital capacity of the lungs. The thorax of the athlete exhibits a power of expansion greatly in excess of that which is possessed by persons who habitually engage in sedentary occupations; and it is only by comparative study of various types of thorax that the examiner may form conclusive opinions as to what degree of thoracic expansion is to be considered abnormal in the individual case under investigation.

Bilateral Variations.—Increased general expansion of the thorax in all of its diameters is observed after active physical effort and during violent emotional excitement without possessing untoward significance. In hypertrophic emphysema, and in the course of a paroxysm of bronchial asthma, as well as in purely costal respiration due to abdominal disease, a general increase in the expansion of the thorax is the rule.

Decreased general expansion of the thorax in its several diameters is characteristic of the small chest of elderly patients with atrophic emphysema. A similar decrease in expansion occurs as a result of general muscular weakness and in paralysis of the intercostal muscles. Obstruction of the upper air passages by limiting the free ingress of air to the lungs is attended by limitation of the thoracic expansion; and bilateral pleurisy with effusion and the bilateral consolidation of double pneumonia result in a similar diminution of the inspiratory excursion of the chest wall. The pain of diaphragmatic pleurisy, pleurodynia, and intercostal neuralgia causes a general diminution of thoracic expansion. Scleroderma affecting the chest wall and premature calcification of the costal cartilages result in general limitation of expansion.

Unilateral Variations.—Increased expansion of one side of the thorax occurs in the presence of vicarious expansion of one lung in compensation for a crippling of its fellow. The cause of the imperfect expansion of the unsound lung may reside in the lung itself in the form of tuberculous or pneumonic consolidation, th collapse of atelectasis, or the fibrosis of chronic interstitial pne monia, fibroid phthisis, or pulmonary syphilis; or, on the co trary, the cause of malfunction may be extraneous to the lun and may assume the form of pulmonary compression by pleur effusion, pneumothorax, or mediastinal tumor.

Decreased expansion of one side of the thorax is noted as consequence of diminution of the air space of the lung by th consolidation of phthisis or pneumonia or the fibrosis of chron interstitial pneumonia, pulmonary syphilis, or fibroid phthisi Stenosis of a principal bronchus, resulting in extensive atelectasi leads to diminution or abolition of expansion upon the corre ponding side of the thorax. Pleural effusion, pneumothorax, tumor of the lung or pleura, impairs the thoracic expansion upo the side of the disease and results in compensatory expansion the opposite side. Unilateral diaphragmatic paralysis, as we as increased subphrenic pressure due to extensive enlargeme of the liver or spleen and the traction of pleural adhesions, cau limitation of thoracic expansion upon one side.

Not infrequently the lesion which is responsible for the d ficient expansion produces deformity of the thorax as well, whi in certain instances is characteristic of the cause. In lar pleural effusion and in pneumothorax, in addition to the unilater immobilization of the thorax, there is unilateral bulging of th chest wall. In limitation of expansion due to pleural adhesio and to advanced phthisis, on the contrary, there is apt to unilateral retraction of the thorax. Moreover, a unilateral r traction which is most marked in, or which is limited to, th upper thorax is suggestive of phthisis, whereas a similar retra tion of the lower anterolateral thorax points rather strongly postpleuritic adhesions as the cause.

Local Variations.—Circumscribed areas of increased thorac expansion are encountered over the apices and the bases of th lungs, and the location of the vicarious expansion in these cas is of potent diagnostic import. Increased expansion over a pu monary apex, limited to the supraclavicular and infraclavicul regions, occurs in the consolidation of lobar pneumonia invol ing the lower lobe of the lung, and in compression of the lun by pleurisy with effusion, mediastinal tumor, or excessively hype trophied heart. In the presence of extensive tuberculous co solidation of the apex, on the contrary, the lower portion of th thorax expands vicariously in compensation for the deficient expansion of the upper lobe of the lung.

Local or circumscribed diminution in the degree of expansion of the thorax at the pulmonary apex in the supraclavicular and infraclavicular regions is noted in apical pulmonary tuberculosis. A similar limitation in expansion of the lower anterolateral regions of the thorax is commonly due to the traction of pleural or of pleuropericardial adhesions.

Wavy expansion of the thorax is occasionally encountered in connection with lobar pneumonia, when successive areas of the thorax appear to expand in an irregular sequence, one area expanding prior to the expansion of adjacent areas.

Inspiratory retraction of the lower intercostal spaces in the lower axillary and infraaxillary regions is a normal phenomenon in the first half of the inspiratory phase of the respiratory cycle. During the first half of the act of inspiration the interspaces are moderately retracted in this area, to become flattened out upon the same plane as the ribs during the second half of the respiratory act, but never exceeding this level throughout the respiratory cycle. Bäumler holds that this initial respiratory retraction of the interspaces in the normal subject is due to the contraction and descent of the diaphragm prior to the contraction of the intercostal muscles, in this wise creating a transitory diminution in intrathoracic pressure or a negative thoracic pressure, with the result that the atmospheric pressure exerted upon the exterior of the thorax during the initial stage of the inspiratory act causes recession of the lower interspaces at this time.

Pathologic inspiratory recession of the lower intercostal spaces differs from this normal inspiratory recession in the fact that the period of the recession consumes the entire time of the act of inspiration. The distribution of the retraction possesses definite localizing value in these cases. As the obvious cause of the retraction in this case is the inability of the lung to become fully inflated with air, if the retraction is bilateral, affecting both lungs to an equal degree, the seat of obstruction is situated above the tracheal bifurcation; whereas, if one entire side of the thorax exhibits pathologic inspiratory retraction of the intercostal spaces, the seat of the stenosis is evidently in one of the primary bronchi. Circumscribed areas of inspiratory retraction signify stenotic lesions of the smaller bronchial tubes.

Local Pulsation

Local areas of pulsation of the thoracic surface possess vari significance, depending upon the character and the location the pulsation. A systolic pulsation immediately above the ba of the heart points to aortic aneurysm. A similar pulsation up the left anterior thorax, between the second and the sixth ri is frequently a sign of pulsating pleurisy. A local, pulsati area overlying the left lung upon the posterior wall of t thorax, below the angle of the left scapula, is occasionally due a large pulmonary cavity containing fluid, to which the imp of the heart is communicated during ventricular systole. A c cumscribed pulsation over the lower anterolateral surface of t thorax, attended by local edema of the chest wall, occurs empyema necessitatis when rupture is imminent.

CHAPTER III

PALPATION

Object and Technic.—Palpation is employed in physical examination of the organs of respiration to confirm the findings upon inspection as to the size and shape of the thorax, the respiratory movements, and degree of expansion of the chest, and to detect slight deficiencies of expansion which are so slight as to have escaped detection during inspection. Palpation is also employed in the detection and analysis of several types of vibration or *fremitus* arising in the thorax during health and in diseased states. The systematic and skillful practice of palpation also reveals the degree of resistance of certain lesions developing within the thorax, as also the presence of local tenderness, local areas of pulsation, and fluctuation. Moreover, palpation yields information as to the tension of the muscular wall of the thorax, the presence of local edema, and the condition of the ribs and the intercostal spaces.

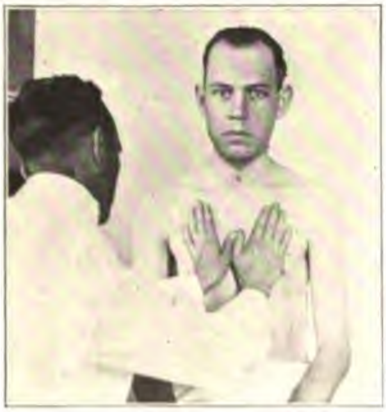


Fig. 30.—Palpation of anterior thoracic surface.

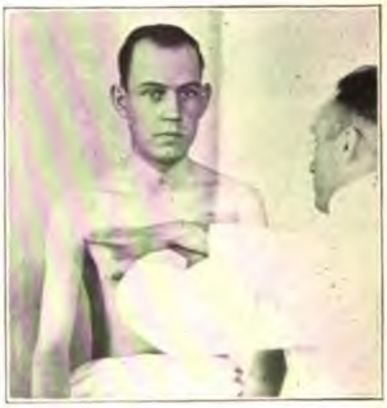


Fig. 31.—Ulnar palpation of thorax.

As employed in the routine physical examination, palpation consists in the application in the first instance of the palmar surfaces of the hands to the surface of the thorax for the purpose of appreciating and analyzing variations in the tactile impressions which are conveyed to the palpating hands. This maneuver is employed in the examination of the anterior, lateral, and po terior regions of the thorax. In palpation of the supraclavicula and infraclavicular regions, on the contrary, as well as in palpa

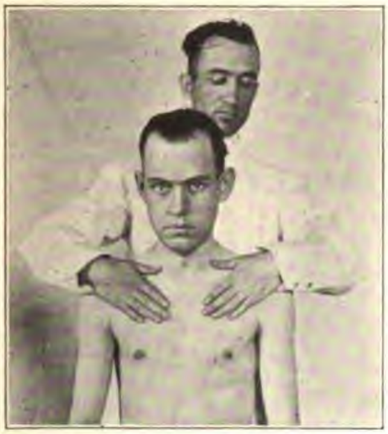


Fig. 32.—Palpation of upper anterior thorax.

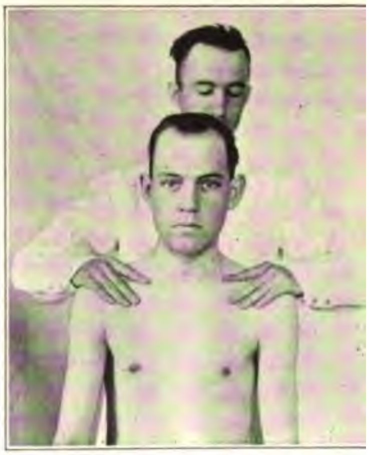


Fig. 33.—Palpation of pulmonary apic

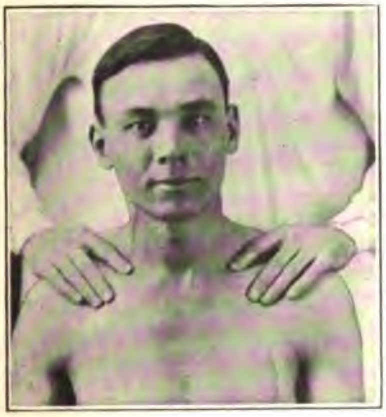


Fig. 34.—Detection of lagging at apices.

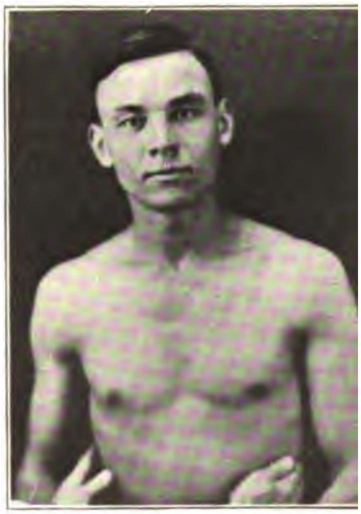


Fig. 35.—Detection of lagging at pulmonary bases.

tion of the intercostal spaces, palpation with the finger-tips alo is more serviceable. Certain clinicians recommend the emplo ment of the ulnar border of the hand in preference to the pal in the practice of palpation; but as a rule, the tactile sensatio which are appreciated by this portion of the hand are not as fine as are those which may be detected by the more sensitive palms.

During palpation of the thorax the clothing should be removed to the waist so that no fabric may intervene between the palms of the examiner and the thorax of the subject.

Palpation may be practiced with the patient in the sitting or recumbent posture, preferably in the former; and during examinations in the recumbent posture it is essential that the patient be turned a sufficient number of times to insure palpation of all portions of the thorax. In either position the patient should be in a state of complete muscular relaxation. The hands of the examiner should be warm, and they should be applied firmly and evenly to the area under examination.

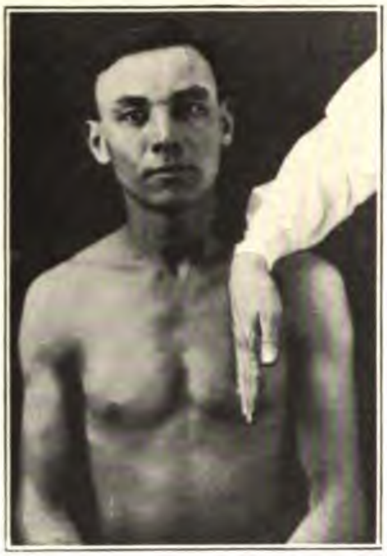


Fig. 36.—Linear palpation of thorax.

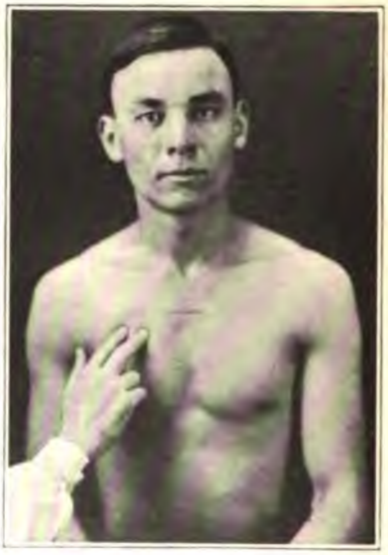


Fig. 37.—Palpation of the intercostal spaces.

In testing the expansion of the pulmonary apices the examiner should stand behind the patient, and with the index and middle fingers in the supraclavicular and infraclavicular regions, respectively, he should estimate the degree of expansion of the apices at the completion of full inspiration. In the detection of deficient expansion of the lateral regions of the thorax the examiner should assume a position in front of the patient and apply the palms of the two hands to the lower lateral regions of the thorax during inspiration. In testing the anter expansion, he should stand beside the patient, and, placing one palm upon the middle of the sternum and the other between the scapulæ, should note the degree of expansion at the completion of inspiration.

Palpation, to be serviceable, must be systematic, the entire surface of the thorax being gone over in orderly succession and corresponding regions upon the two sides should be compared. Hasty and unsystematic palpation is a frequent source of erroneous conclusions.

THORACIC VIBRATIONS

Palpable vibration of the thoracic wall (fremitus) is produced by the vibrations of the vocal cords during phonation, by the movement of the roughened surfaces of an inflamed pleura, by the action of coughing, by the forcible movement of fluid in the pleural cavity, and during the percussion of the thorax in the presence of hydatid cyst. These various forms of tactile fremitus are manifested in characteristic manner, and each form furnishes valuable data upon which the examiner may base conclusions.

VOCAL FREMITUS

Vocal fremitus is manifested in the form of a palpable vibration of the thoracic wall, which is conveyed to the palms when these are applied to the surface of the thorax during phonation. The vibration, which has been likened to the sensation which one experiences upon placing the hand upon a resonating box in which a tightly stretched wire or cord is caused to vibrate rapidly, is appreciated immediately upon the act of phonation. Its duration corresponds almost, but not quite, exactly to the duration of the spoken words, the tactile fremitus being prolonged during a very brief space of time after the cessation of speech on the part of the subject.

These vibrations, which originate in the vocal cords of the larynx, and which are the physical basis of the production of the voice, are communicated directly to the two columns of air situated respectively above and below these cords. The aerial column situated below the true vocal cords constitutes a continuous conducting medium throughout the larynx, trachea, bronchi, bronchioles, and pulmonary alveoli, a medium which thus extends to periphery of the lungs. From the pulmonary alveoli the ibrations are conducted by way of the alveolar walls and the investing pleura to the thoracic walls, and by these walls to the palpating hand placed upon the surface of the thorax.

As the air passages form a closed system, the vibrations arising in the vocal cords are naturally conducted downward, since the lateral propagation of the waves is effectually prevented by the walls of the tubes. Doubtless the solid walls of the air passages are also concerned to a minor degree in the conduction of the vocal vibrations along their course to the thoracic wall; but in this function these structures can play only a minor rôle, in accordance with the physical principle that the transmission of vibrations along a solid structure is enfeebled in direct proportion to the degree of variation in the structure of the conducting medium. Certainly the waves of vibration encounter numerous adverse changes of structure in the course of their conduction by way of the solid structures from their origin in the larynx to the termination of the finer bronchioles in the periphery of the lung.

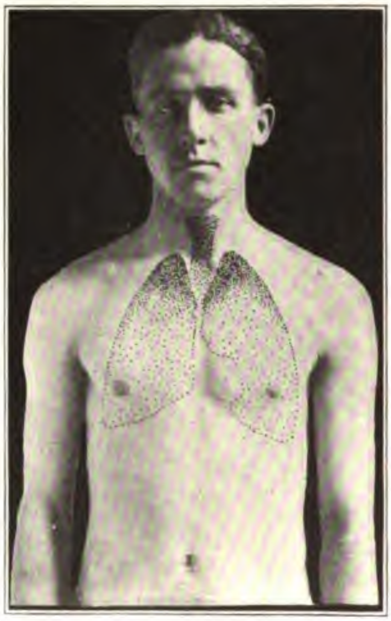


Fig. 38.—Normal variations in vocal fremitus.

In eliciting vocal fremitus the examiner should apply the palms or the ulnar borders of the hands to the surface of the thorax while the patient is directed to count "one, two, three," or to repeat the words "ninety-nine" in a deep voice of uniform intensity, the examiner meanwhile noting the intensity with which the vibrations are transmitted to the palpating hand in the various regions of the thorax.

The intensity of vocal fremitus in the normal subject is governed by the intensity of the voice during phonation; by the pitch of the speaking voice; by the variations in the relations of the primary bronchi to the thoracic wall; and by the varying thickness of the thoracic wall in different regions of the thorax.

The intensity of vocal fremitus varies directly with the intensity of the speaking voice. Just as the voice is stronger in proportion as the amplitude of each vibration is greater, so also are vocal vibrations of greater amplitude manifested by tactile vibrations of corresponding intensity. If, during palpation of the thorax, the patient is directed to speak continuously in a voice of progressively increasing intensity, the resulting vocal fremitus will be observed to become progressively more intense. If, on the contrary, the speaking voice is so modulated as to become progressively less intense, there is witnessed a corresponding diminution in the degree of tactile fremitus, which indeed is
pletely abolished when the voice is reduced to a whisper.
of the first importance during the elicitation of vocal

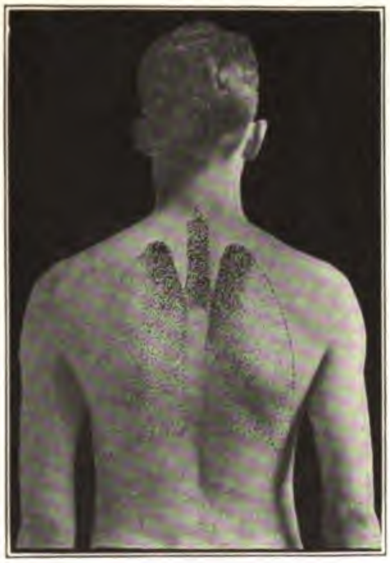


Fig. 39.—Normal variations in vocal fremitus.

fremitus for diagnostic purposes to see that the subject speaks in a voice of uniform intensity throughout the examination.

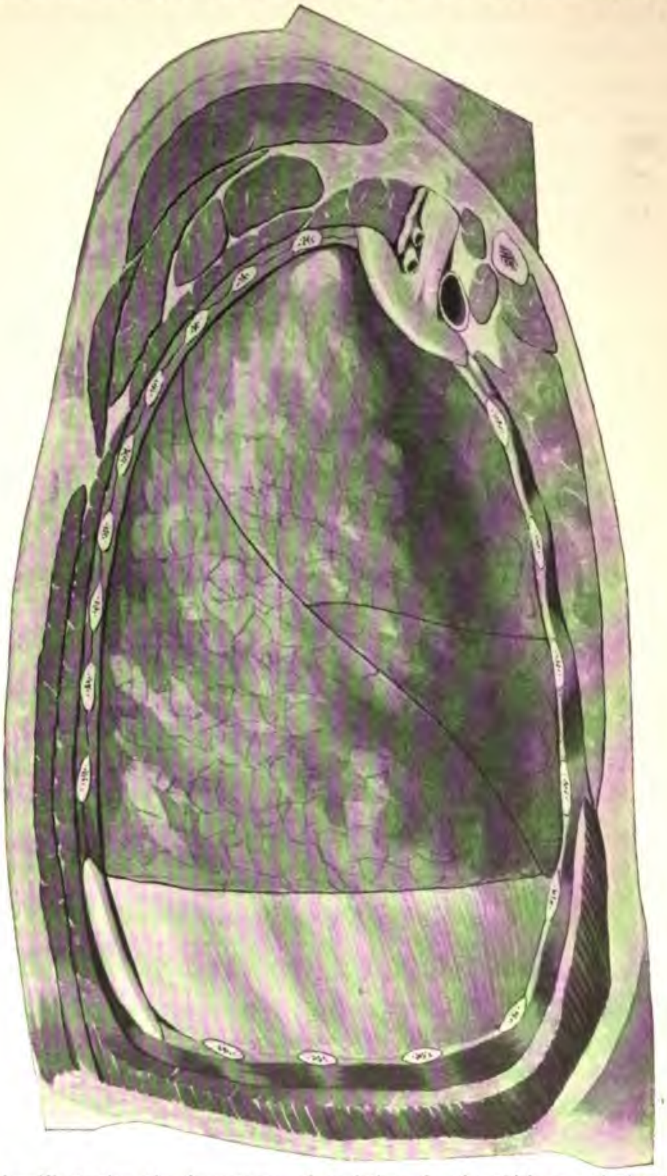


Fig. 40-A.—Illustrating the importance of variations in the thickness of the thoracic wall upon the interpretation of physical findings upon palpation of the thorax.

The pitch of the voice exerts a striking influence upon the intensity of vocal fremitus, the tactile vibration being most intense in subjects with deep, bass voices, and possessing the min-

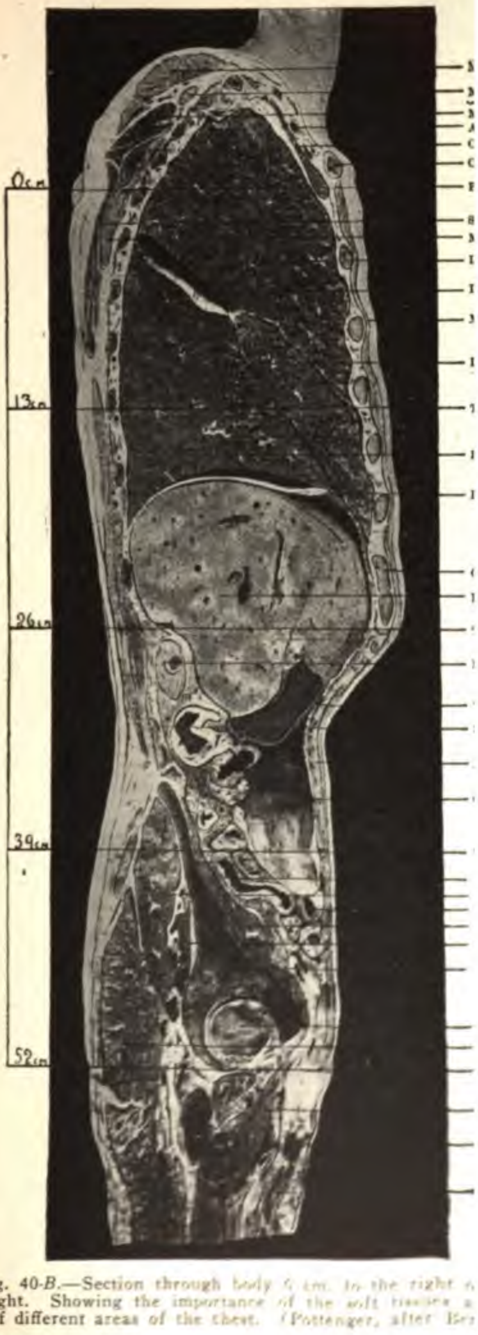


Fig. 40-*B*.—Section through body 6 cm. to the right o
the right. Showing the importance of the soft tissues a
tion of different areas of the chest. (Pottenger, after Be

imum of intensity in subjects of high suprano voices. Indeed if, in a suitable subject, the examiner will palpate the thorax during phonation of progressively ascending pitch, in the case of the high suprano voice, a point will be reached at which the tactile fremitus disappears. The underlying physical basis of this progressive enfeeblement of vocal fremitus resides in the fact that as the pitch of the voice ascends, the sound waves become progressively more rapid with a corresponding diminution in their amplitude, until the point is reached at which palpable vibration of the thoracic wall is no longer produced. Upon these physical principles it is readily understood that vocal fremitus is normally more intense in the adult male subject with his naturally deep voice called forth by waves of moderate rate and wide amplitude; and that it is correspondingly less intense in the adult female subject and in children, in whom the voice possesses less volume but gains in pitch, or, in other words, in whom the sound waves possess a more limited amplitude but a higher rate of vibration.

Vocal fremitus in the normal subject is slightly more intense upon the right side of the thorax than upon the opposite side, because of the greater caliber of the right bronchus and because of its anatomical situation in closer proximity to the anterior thoracic wall than that of the left bronchus. It follows that during phonation a more considerable column of air is set in motion in the right bronchus, and its closer relation to the thoracic wall further facilitates the transmission of the vibrations to the palpating hand. Vocal fremitus is naturally most pronounced in those regions of the thorax where the larger bronchi approach the thoracic parietes; and it exhibits impairment in those areas where the bronchi are separated from the chest wall by the intervention of aerated pulmonary tissue. Hence, it presents its maximum intensity in the root of the neck, immediately over the course of the larynx and the trachea, and is more intense over the right apex than upon the corresponding area of the opposite side.

The condition of the thoracic wall exercises a considerable influence upon the intensity of vocal fremitus. The deep, muscular thorax of the robust subject and the thorax which is clothed with a thick paniculus adiposus in the obese subject present a striking enfeeblement of the vocal vibrations; whereas these are unduly intense upon palpation of the thin, poorly muscled thorax of the emaciated subject, as well as in the thin

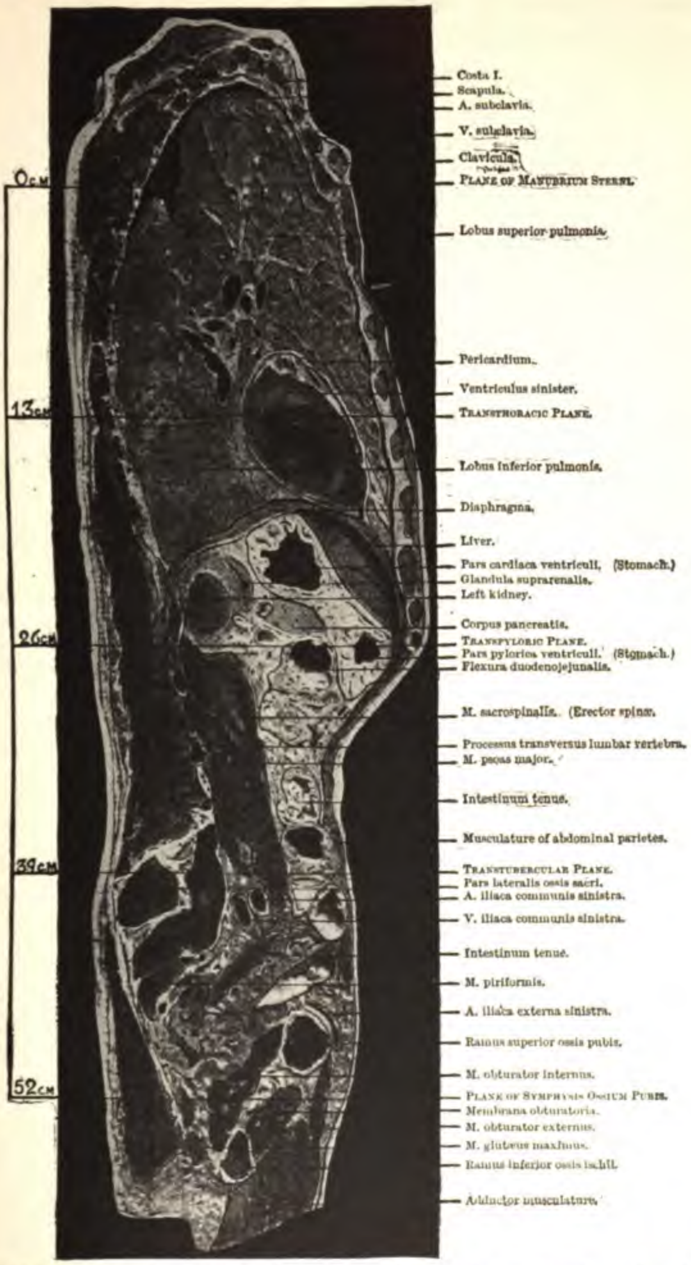


Fig. 40-*C*.—Section through body 6 cm. to the left of the median plane viewed fro
the right. Showing the importance of the soft tissues as influencing physical examin
tion of different areas of the chest. (Pottenger, after Berry.)

and elastic thorax of the female subject and the child. Extensive edema and local suppuration of the thoracic wall similarly mask the integrity of the tactile vibrations. Upon more minute examination, it can usually be demonstrated that vocal fremitus is normally more intense over the intercostal spaces than it is in the regions which overlie the ribs. It is very natural that the intercostal musculature should respond more readily to the intrathoracic vibrations than the less elastic osseous ribs.

Vocal fremitus presents certain *regional variations* in the intensity with which it is transmitted to the palpating hand in various regions of the thorax. Considering the thorax as a whole, the fremitus in the normal subject is most intense upon the anterior thoracic wall; it is slightly less intense upon the lateral walls; and it exhibits its minimum of intensity upon the posterior wall of the thorax. A number of variations in the relative intensity of the fremitus in the normal subject are to be encountered upon each of these three aspects of the thorax.

Upon the anterior surface of the thorax the fremitus is less intense in the supraclavicular region, which corresponds to the anterior surface of the apex of the lung, than upon any other portion of this surface with the exception of the narrow zone immediately overlying the bony clavicle and over the distribution of the mammary gland in the female subject. In the supraclavicular region the fremitus becomes progressively more intense as the examiner palpates inward toward the median line, owing to the proximity of the trachea to the inner portion of this region.

Upon palpation of the narrow zone immediately superjacent to the clavicle, the tactile fremitus is greatly enfeebled, contrasting in a striking manner with the more pronounced vibrations which are encountered in the supra- and infra-clavicular regions above and below the bone. But even this limited area of the thoracic surface presents certain variations in the intensity of the fremitus, which exhibits its greatest strength upon palpation of the inner third of the clavicle, near its junction with the sternum. In the middle third of the bone the fremitus exhibits a moderate diminution over that of the inner third, a diminution which progressively increases in the outer third of the bone as the palpating hand approaches the scapula.

Upon passing from the clavicular area to the infra-clavicular region, there is a very appreciable reinforcement of the fremitus. This intensity is moderately reduced in the mammary region in the male subject, and quite markedly reduced over the mammary gland in the female subject.

In the areas in which the heart and the liver come into direct contact with the anterior thoracic wall there is an abrupt cessation of vocal fremitus. Linear palpation with the ulnar border of the hand is very serviceable in accurately determining the precise points at which the fremitus terminates in relation to these organs.

In the median line of the thorax, in the sternal region, at least three variations in the intensity of vocal fremitus may be detected. In this region the fremitus is most feeble over the upper portion, which corresponds anatomically to the manubrium sterni; it attains its maximum strength over the gladiolus; and it is again enfeebled in the lower portion of the region, which overlies the ensiform cartilage.

In the ventral aspect of the neck, in the region in the median line which overlies the larynx and trachea, vocal fremitus is elicited in its purest form. Even in this restricted region, however, certain variations in its intensity may be appreciated by careful palpation. Its point of maximum intensity corresponds to the level of the inferior border of the thyroid cartilage, from which point the vibrations become progressively enfeebled as the palpating hand travels upward toward the floor of the mouth and downward toward the episternal notch.

Upon the lateral thoracic walls vocal fremitus is most intense in the upper portion of the axillary region, becoming progressively more feeble as one palpates downward over the lower axillary and infraaxillary regions. Upon the right side of the thorax the fremitus is interrupted abruptly at the upper border of the eighth rib by the apposition of the liver with the thoracic wall. Similarly, upon the left lateral aspect of the thorax the fremitus is interrupted at the upper border of the ninth rib by the apposition of the spleen with the thoracic wall.

Upon the posterior wall of the thorax vocal fremitus presents its greatest intensity in the interscapular regions, overlying the trachea and its bifurcation to form the two primary bronchi. In the subscapular regions, overlying the pulmonary bases, the fremitus undergoes a considerable diminution in intensity by reason of the considerable distance of these regions from the site of production of the vocal vibrations. In the regions overlying the scapulæ the fremitus is more feeble than elsewhere upon the posterior thoracic wall, the point of minimum intensity corresponding accurately to the course of the spine of the scapula.

PATHOLOGIC VARIATIONS

In the presence of disease of the respiratory organs numerous gradations in the intensity of vocal fremitus are encountered. As a result of morbid changes in the bronchi, lungs, pleura, or thoracic wall, the fremitus may be found to be increased in intensity, to be diminished in intensity, or to be entirely abolished over certain regions of the thorax. In general, affections of the pleura are attended by diminution of vocal fremitus, while disease of the lungs is manifested by exaggeration of the fremitus; but in the interpretation of the findings in any case it is to be remembered that affections of the bronchi and of the thoracic wall are capable of causing either diminution or reinforcement of vocal fremitus independently of disease of the lung or the pleura.

Increased Vocal Fremitus.—Vocal fremitus is increased in intensity in all conditions which are attended by solidification of the pulmonary tissues with a concomitant diminution in the air content of the area in question. This condition of solidification and anaeration obtains in the consolidations of pneumonia, phthisis, and hemorrhagic infarction of the lung; in the presence of diffuse carcinomatous infiltration of the lung, provided that this infiltration does not cause bronchial occlusion; in pulmonary atelectasis from external pressure; and in indurative contraction of the lung from fibroid phthisis, chronic interstitial pneumonia, or pulmonary syphilis. It is essential, however, for consolidations to yield exaggerated tactile fremitus, that the consolidated areas be situated in the periphery of the lung, and that the bronchi which terminate in the consolidated areas be free from occlusion.

The physical basis for the exaggeration of vocal fremitus under these circumstances is readily appreciated, since a continuous medium of uniform structure conducts vibrations with far greater intensity than does a conducting medium which is constantly undergoing variations in structure, as are the normal pulmonary alveoli, in which thin walls of pulmonary tissue constantly are interrupted by minute air-containing spaces. When a solid neoplasm of the lung occupies the peripheral portion of this organ and at the same time extends so deeply into the lung as to come into contact with one or more of the larger bronchi, the physical conditions are eminently favorable for the transmission of vocal fremitus to the surface of the thorax with the maximum degree of exaggeration.

In the presence of compression of the pulmonary tissues by a pleural effusion, the exaggeration of vocal fremitus is ordinarily suppressed over the lower portion of the thorax, corresponding to the area occupied by the effusion, only to be increased in intensity in the region immediately above the level of the effusion, as a result of compression and relaxation of the pulmonary tissues in the upper portion of the pleural cavity. It is quite otherwise in the case of pulmonary compression by an extensive pericardial effusion. In this instance the compression operates upon the internal surface of the inferior lobe of the left lung, leading to exaggeration of vocal fremitus over the compressed lobe. Excessive abdominal distention in ascites, meteorism, or large abdominal tumor, can operate in a similar manner, and exhibit exaggeration of vocal fremitus over the lower segment of the thorax.

Pulmonary cavities with free communication with a bronchus, when they are situated in the periphery of the lung, are productive of exquisite refinements of vocal fremitus. In this case the cavity acts as a resonating chamber for the amplification of the vocal vibrations. It is essential, however, that the cavity in these cases be free from fluid secretion, which may obstruct the bronchial orifice and consequently suppress vocal fremitus over its distribution. Bronchiectases, when peripherally distributed, cause similar exaggeration of tactile fremitus, bronchiectasis being the sole affection of the bronchi which is capable of increasing the intensity of vocal fremitus.

Diminished Vocal Fremitus.—Partial stenosis of the bronchi by tenacious secretions in the course of chronic bronchitis, or stenosis arising as a consequence of the lodgment of foreign bodies, or of bronchial compression by cicatricial bands, aneurysm, or extensive pericardial effusion, results in a diminution of vocal fremitus over the ultimate distribution of the affected tubes. When it is a case of partial occlusion by accumulated secretions, the fremitus is occasionally restored in its integrity after a severe paroxysm of cough.

Pleural thickening, sometimes noted at one apex as a result of chronic tonsillar infection, as well as in other portions of the pleural membrane as a sequel of pleural inflammation, is an occasional cause of diminution of vocal fremitus. That pleural thickening is not invariably attended by diminution of vocal fremitus was conclusively demonstrated by Wintrich in his experiments upon the cadaver. In his experiments he demonstrated that upon investing the exposed pleura with membranes prepared from the stomach or intestine there was no appreciable change in the intensity of the vocal vibrations when an assistant spoke into a tube which was inserted into the primary bronchus of the corresponding lung.

A very important group of cases, a group which is often quite confusing to the examiner at the bedside, is composed of certain cases of pulmonary infiltration and consolidation which, instead of yielding exaggeration of vocal fremitus upon palpation, exhibit an enfeeblement or abolition of tactile vibrations upon palpation. This condition is encountered in massive pneumonia, in which the terminal bronchi become occluded by fibrinous exudate with consequent suppression of vocal fremitus, and strongly simulating in this respect the picture of extensive pleural effusion. With less frequency, in the presence of carcinomatous infiltration of the lung, the neoplastic new productions develop in the interior of the bronchi, causing partial or complete occlusion of these passages, with consequent diminution or abolition of vocal fremitus over the area of the thorax corresponding to their distribution.

When the lung is separated from the thoracic wall by pleural effusion or by the accumulation of air or gas in the pleural cavity, vocal fremitus is enfeebled or is abolished. In the case of pleural effusion, it is possible for extensive adhesions between the visceral and the parietal pleura to transmit the vibrations across the effusion; and in these cases, also, the fremitus is ordinarily accentuated over the portion of the lung which is compressed into the superior part of the pleural cavity above the level of the effusion.

It is desirable, but not always possible, in connection with a pleural effusion to determine whether the presence of the fluid in the pleural cavity is the sole cause of diminution of the fremitus. Occasionally compression of the lung by the effusion results in compression and occlusion of the larger bronchi, a factor which plays an important part in suppressing the vocal fremitus. Moreover, in the presence of extensive effusions the thoracic wall is in a state of abnormal tension, which interferes with the transmission of vibrations arising within the thorax.

In dealing with pleural effusions, if the upper limit of the area of impaired vocal fremitus is carefully marked upon the thorax, the examiner is enabled to judge, during consecutive examinations, of the progress of the malady. In this disease, however, a sudden and pronounced descent of the level of the fluid is more apt to be due to relaxation of the diaphragm upon the affected side than to a sudden and pronounced resorption of the fluid.

In the case of encysted or loculated pleurisy, the examiner may occasionally delimit the effusion with fair accuracy by means of careful palpation with the ulnar border of the hand. However, Jaccoud, who has studied multilocular pleurisies with special reference to the variations in vocal fremitus, warns the examiner against the possibility of error in reaching a diagnosis of encysted pleurisy through the findings of palpation alone.

The accumulation of gas or air in the pleural cavity is attended by diminution of vocal fremitus over its distribution. In the case of pneumothorax limited to the apex of the pleural cavity, which yields a tympanitic note upon percussion, the enfeeblement or abolition of vocal fremitus over this area is of considerable aid in the differential diagnosis between a gaseous accumulation and a large tuberculous cavity at the apex of the lung.

In the interpretation of data obtained by the study of variations in the intensity of vocal fremitus in the various regions of the thorax, it is essential that the examiner be constantly upon the alert to detect any changes in the thoracic wall which might influence the intensity of the fremitus as appreciated by the palpating hand. In the presence of atrophy of one great pectoral muscle or of the muscles of the shoulder girdle, there is a corresponding exaggeration of vocal fremitus in the corresponding region, which might, during a casual examination, suggest the presence of apical consolidation. Circumscribed areas of edema of the thoracic wall and the presence of peripleuritic suppuration, on the other hand, are capable of producing a circumscribed diminution of vocal fremitus in the absence of bronchial or pulmonary disease. In the thorax which is the seat of extensive deformity from osseous or pulmonary disease deductions should be drawn from variations in vocal fremitus with caution and circumspection.

Absence of Vocal Fremitus.—All of the pathologic conditions which are capable in the course of pulmonary disease of abolishing vocal fremitus have been enumerated in the foregoing paragraphs. The fremitus is abolished over the circumscribed area of the thoracic surface which corresponds to a large pulmonary or bronchiectatic cavity which is filled with fluid secretions which deprive the cavity of aerial content and occlude the communicating bronchus. A similar circumscribed area of abolished fremitus is occasionally encountered at the level of an aortic aneurysm or over a solid tumor of the lung or pleura which is not in intimate relation with a bronchus.

Vocal fremitus is abolished over an extensive area of the lower segments of the thorax in the presence of massive pleural effusion; and over areas of the lung in which the bronchus supplying the part has become completely obstructed from any cause. Also, in massive pneumonia vocal fremitus may be abolished over the area of consolidation as the result of obstruction of the bronchioles and bronchi by fibrinous plugs.

RHONCHAL FREMITUS

The term rhonchal fremitus, or bronchial fremitus, was first applied by Guttman to the vibrations which are generated by the passage of air in the respiratory passages through serum, mucus, pus, or blood in the bronchial tubes. The vibrations which are thus produced are readily appreciated by the palpating hand applied to the thorax. The lesions which are ordinarily responsible for the production of rhonchal fremitus are situated in the larger and medium-sized bronchial tubes when it is a question of the passage of air through tenaceous secretions; but the lesions which are provocative of the fremitus also not infrequently assume the form of tuberculous cavities containing fluid with a bronchial communication situated below the level of the fluid, through which air ascends during inspiration and creates bubbling sounds which are transmitted to the thoracic surface in the form of rhonchal fremitus. Hence, it follows that rhonchal fremitus may possess a variable diagnostic and prognostic significance depending upon the mechanism of its production and the region of the thoracic surface upon which it is encountered.

The rhonchal fremitus which originates in the larger and medium-sized bronchi in the course of chronic bronchitis and bronchial asthma, and which is not of especially grave prognostic import, is encountered in the upper sternal, lower infraclavicular, and upper mammary regions. Rhonchal fremitus encountered in these regions has a very considerable intensity, and it is commonly felt over a considerable area of the thoracic surface. Rhonchal fremitus which is engendered in a tuberculous cavity, on the contrary, is ordinarily encountered in the supraclavicular and infraclavicular regions, corresponding to the apices of the lungs; it has not the intensity of the fremitus generated in the larger bronchi; and it is of grave prognostic import. In palpating in these regions, the examiner will not infrequently encounter crepitations which are due to the contractions of the great pectoral muscle, particularly when dealing with robust subjects, and which may easily be mistaken for rhonchal fremitus.

Similarly, rhonchal fremitus is apt to be confused with pleural friction fremitus. In this connection it is to be remembered that in dealing with pleural friction fremitus, the latter is exaggerated upon compression of the intercostal spaces; that pleural friction fremitus is ordinarily attended by pain which is exaggerated by pressure exerted with the finger-tips in the intercostal spaces; and that rhonchal fremitus arising in the bronchial tubes is frequently abolished by the act of coughing, whereas pleural friction fremitus is not influenced by this act.

Rhonchal fremitus is transmitted to the thoracic surface with the maximum intensity in women and children, in whom the thoracic wall is thin and elastic, and in the emaciated patient; and due allowance should be made for the relative thickness of the thoracic parietes in interpreting the intensity of rhonchal fremitus in any case in which it is encountered.

PLEURAL FRICTION FREMITUS

In the normal subject the visceral and the parietal pleura are moistened by a small quantity of serous fluid which enables the membranes to glide over each other during the respiratory movements of the lungs and the thoracic walls without the production of sound. In the presence of pleural inflammation, however, the surface of the membrane becomes invested by a coating of fibrinous exudate of varying thickness, by virtue of which there is produced during the respiratory movements an audible rubbing sound, the pleural friction sound. Upon palpation of the thorax over the region of production of the pleural friction sound there is frequently encountered a palpable vibration or fremitus, which Guttman first designated as pleural friction fremitus. The tactile impression which one obtains is that of grating, which is variable in intensity in different cases and which is dependent upon the respiratory excursions for its generation. It is most frequently encountered in the infraaxillary region and the lower mammary region; and it is most frequently generated in a vertical direction, with less frequency in an oblique direction, and still less frequently in a horizontal direction.

The fremitus is appreciated most frequently during inspiration.

with less frequency during inspiration and expiration, and very rarely during expiration alone. It is not infrequent that the intensity of the fremitus is so slight that it is not perceived during tranquil respiration, but is brought to the fore by a series of deep inspirations. Also, it is not infrequent for the fremitus to disappear after a series of deep inspirations, probably by reason of the smoothing out upon the pleural surface of the rugosities of exudate which called the fremitus into being. At any rate, the fremitus frequently reappears upon deep inspiration following a period of tranquil respiration on the part of the patient. The intensity of the fremitus can be reinforced by pressure upon the intercostal spaces in the area of its production, a procedure which is attended by exaggeration of the thoracic pain.

Pleural friction fremitus is encountered in the presence of acute fibrinous pleurisy and during the incipient stage of serofibrinous pleurisy. In the latter disease the fremitus is present prior to the development of the effusion; it disappears with the free establishment of effusion; and it very frequently recurs with partial or complete absorption or evacuation of the effusion.

TUSSILE FREMITUS

Tussile or tussive fremitus is a palpable thoracic vibration which is produced upon coughing. It is not invariably present; it does not possess great value in physical examination; and it is not frequently elicited during the routine physical examination. It is to be employed in dealing with patients who are subject to aphonia from any cause, when it is not possible to elicit vocal fremitus.

SUCCUSSION FREMITUS

Succussion fremitus is a palpable thoracic vibration which is produced when a patient whose pleural cavity contains air and fluid is suddenly jarred or shaken. Under these circumstances the impact of the fluid is felt against the palpating hand as a vibration.

Occasionally extensive pulmonary cavities which are situated in the peripheral portions of the lung give the vibration, if they contain air and fluid. However, the physical conditions are not ideal for the elicitation of this sign from pulmonary cavities; and it is much more frequently encountered in hydro-, hemo-, or pyo-pneumothorax.

HYDATID FREMITUS

In the presence of hydatid cyst of the lung with visible protrusion of the thoracic wall, it is occasionally possible to elicit hydatid fremitus by placing the fingers, widely separated upon the bulging area and percussing lightly upon one of the fingers. Under these circumstances a vibration which is caused by the impact of the daughter cysts is occasionally appreciated by the fingers.

CREPITATION

In cases of surgical emphysema, when the subcutaneous tissues of the thorax contain small beads of air, a fine crepitation is often demonstrable upon palpation of the affected area with the finger-tips. Air may gain access to the subcutaneous tissues of the thorax as a result of trauma or operation upon the neck or chest, or from the rupture of dilated infundibula in hypertrophic emphysema.

LOCAL TENDERNESS

In the presence of disease of the pleura and in disease of the lung complicated with pleurisy, palpation frequently reveals areas of local tenderness upon the thoracic surface. The pain in these cases is elicited and is also defined by finger-tip palpation of the intercostal spaces. Such localized tenderness is suggestive, in the first place, of acute fibrinous pleurisy; and with less constancy it points to intercostal neuralgia, pleurodynia, fracture or caries of the ribs, deeply seated disease of the lung, or disease of abdominal organs.

Bau and Bouillaud explain the local sensibility of certain intercostal spaces in connection with acute fibrinous pleurisy on the basis of a neuritis of the corresponding intercostal nerves. They call attention to the fact that the intercostal nerves in the posterior third of their course are in direct relation with the under surface of the pleural membrane, and Bau has demonstrated microscopically that neuritis occurs in connection with this pain in association with acute fibrinous pleurisy and pleuro-pneumonia.

The fact that the pleural pain is manifested not upon the posterior thoracic surface, but upon the lateral and anterior aspects of the chest is explained by the fact that irritation of the nerve trunk is transmitted throughout its terminal expansions. The predominance of the pleural sensibility in the sixth and seventh intercostal spaces is due to the fact that this region of the thorax is more mobile than other portions, and the extreme mobility of these interspaces intensifies the pain upon pressure with the finger-tips.

Malloizel, who has studied pleural pain with special reference to interlobar pleurisies, was able in many instances of this disease to elicit tenderness which corresponded accurately to the course of the interlobar fissure, commencing in the neighborhood of the third dorsal vertebra, and radiating toward the lateral and anterior thoracic regions along the course of the interlobar fissure in question.

While as a general rule even extensive inflammatory disease of the lung is unattended by pain upon pressure exerted in the intercostal spaces, in pleural inflammation of whatever intensity this local tenderness is almost invariably present. However, in the case of lobar pneumonia, Hutinel and Paisseau have described a submammary painful point in adults, whereas in infants and children the pain is commonly referred to the right iliac region, simulating that of acute appendicitis in this class of patients.

The pain of intercostal neuralgia is not infrequently limited to a single intercostal space, but in this space it commonly involves the entire area from the vertebral column to the sternum. Moreover, it is attended by the three painful points of Valleix, pressure upon which accentuates the pain. These points correspond to the points of emergence of the cutaneous filaments of the intercostal nerves. One of these points is situated in the posterior extremity of the intercostal space close to the vertebral column; another is situated upon the lateral thoracic wall equi-distant from the vertebral column and the sternum; while the third is situated upon the anterior thoracic surface adjacent to the lateral sternal border. In addition to the presence of these painful points, the pain of intercostal neuralgia is paroxysmal in its character.

Neuralgia of the phrenic nerve causes characteristic tenderness of the thorax. In this condition pain is elicited upon the exertion of pressure upon the antero-lateral thoracic wall at the insertions of the digitations of the diaphragm into the seventh, eighth, ninth, and tenth ribs; upon the insertion of the muscle to the twelfth rib adjacent to the vertebral column; and upon the spinous processes of the third and fourth cervical vertebræ, which correspond to the origin of the cervical plexus. While neuralgia of this nerve occasionally develops without apparent cause, in many instances the underlying factor is a diaphragmatic pleurisy or a pericardial effusion.

The pain of pleurodynia is most frequently observed upon the left side of the thorax, involving usually the intercostal muscles, and with less frequency the pectoralis major and the serratus magnus. Pressure exerted with the finger-tips, movements of the thoracic wall during respiration, and bending and turning of the trunk intensify the pain. The distribution of the pain is more diffuse than is the case with intercostal neuralgia, and the pain is constant and not paroxysmal as is the case with neuralgia of the intercostal nerves. When pleurodynia involves the pectoralis major muscle the pain is greatly aggravated when the examiner compresses the body of the muscle between the fingers.

In periostitis or caries of a rib, the tenderness is limited to the rib in question, and usually to a single portion of the rib, while compression of the adjacent intercostal spaces is almost devoid of pain. Occasionally there is local bulging and discoloration of the integument over the site of the lesion. Localized pain upon pressure upon a rib is frequently encountered as a result of fracture of a rib.

Local tenderness due to peripleuritic abscess is commonly attended by a local bulging of the thoracic wall, and in the later stages of the condition, when perforation of the chest wall is imminent, there is local bulging and discoloration of the integument.

THE INTERCOSTAL SPACES

In addition to the elicitation of local tenderness, palpation of the interspaces is employed in the estimation of their relative width in cases of pleurisy with effusion, chronic ulcerative phthisis, and in unilateral and local deformities of the thorax due to pulmonary fibrosis or extensive bronchiectasis. In the presence of pleurisy with effusion, the consolidations of lobar pneumonia and phthisis, and in cases of excessive pleural thickening, as well as in fully developed cases of hypertrophic emphysema, finger-tip palpation of the intercostal spaces yields a sensation of increased resistance.

THE RIBS AND STERNUM

In addition to the elicitation of tenderness in the presence of inflammation and caries of the ribs, palpation of these structures in syphilitic subjects occasionally reveals the presence of gummatous nodules. In rickets the rachitic rosary is often present in the form of a series of palpable or even visible nodes occurring in series upon either side of the thorax at the junction of the ribs with their costal cartilages.

Palpation of the sternum is serviceable in the detection of minor variations in the prominence of the angulus Ludovici, and also frequently reveals the presence of caries of this bone. Painful enlargement of the sternoclavicular articulation is more common in connection with gonorrheal rheumatism than in any other disease. Prominence of the superior portion of the sternal region, attended by pain of a dull, boring character, is significant of aneurysm of the aortic arch.

LOCAL PULSATION

Palpation is serviceable in confirming the findings of inspection as to pulsation of various regions of the thoracic surface; for detecting minor degrees of thoracic pulsation which may have escaped detection during inspection; and to gauge the force or lifting power of a thoracic pulsation.

In empyema, when this involves the left pleural cavity, the impact of the heart is communicated to the fluid during cardiac systole, creating thus a systolic pulsation in the lower left anterolateral thoracic region. Frantzel has described a similar systolic pulsation which was transmitted to a purely serous pleural effusion, and Eichhorst has encountered in the child similar pulsations in serous effusions of the right pleura, in this instance localised to the right lower anterolateral thoracic region. Similarly, when the lingula of the left lung which overlies the heart in the left mammary region becomes the seat of dense consolidation in pneumonia or is invaded by dense carcinomatous infiltration, there is generated a systolic pulsation or pulsion of this region of the thoracic wall with each ventricular systole.

In the presence of empyema necessitatis of the left anterolateral thoracic wall, there is produced, just as in the case of aortic aneurysm, a systolic pulsation which is expansile in character, occurring in all directions. This expansile pulsation of the protrusion may be determined with ease by applying the fingers widely separated to the various walls of the sac simultaneously, whereupon the fingers will be moved apart with each systole of the heart which is transmitted to the contained fluid.

In the differentiation of such a pulsating empyema necessitatis from aneurysm Müller calls attention to the fact that empyema is situated in the lower left antero-lateral region of the thorax, whereas aneurysm is localized in the upper and right portion of the thorax; that in the case of empyema, progressive, gentle pressure exerted upon the protrusion will cause its reduction by evacuation of the purulent contents into the pleural cavity through the fistulous communication between the pus sac and this cavity; that in empyema the zone of dullness extends well beyond the limits of the tumor; and that in empyema no systolic bruit is audible as is the case with aneurysm.

A peripleuritic abscess overlying the ventricle of the heart is attended by a systolic pulsation in this area; but in this instance, as the purulent accumulation is enclosed in a cavity with relatively dense walls, the pulsation is manifested as a simple raising and recession and is not expansile.

A palpable pulsation occurring over a relatively broad area upon the left side of the posterior wall of the thorax below the angle of the scapula, is occasionally noted in connection with an extensive tuberculous excavation of the left lung, when this cavity is filled with fluid, to which the impact of the heart is transmitted during systole.

FLUCTUATION

Fluctuation is not ordinarily demonstrable in connection with disease of the thoracic viscera, on account of the rigidity of the thoracic wall. Pleural effusion, if it is very extensive and is attended by marked widening of the intercostal spaces, occasionally yields fluctuation upon tapping the thoracic wall while the fingers of the opposite hand are pressed firmly into the intercostal space. This fluctuation is more frequently demonstrable in the case of children with empyema.

In cases of empyema necessitatis, in which the accumulation of purulent material is extensive and rupture is imminent, pitting upon pressure is readily obtained, and fluctuation is not infrequent. It is elicited by placing the palm of the palpating hand over the protruding area and rather forcibly striking the opposite side of the thorax with the free hand, a procedure which is not devoid of danger in all cases. In most cases of empyema necessitatis the local bulging may be made to disappear by pro- ssive pressure exerted upon the tumor by the palm of the hand. Upon removal of the pressure the tumor resumes its former dimensions. Not infrequently the size of the protrusion caused by empyema necessitatis is variable, depending upon the depth of the respirations. Fluctuation is occasionally demonstrable in the presence of abscess of the pectoral wall, whether due to disease of the thoracic musculature, the ribs, or caries of the bodies of the dorsal vertebræ. Aneurysm of the aortic arch, with erosion of the sternum, presents an expansile, fluctuating tumor, above the base of the heart.

CHAPTER IV

PERCUSSION

Object and Technic.—Percussion was first applied to the exploration of pathologic states of the respiratory organs by Auenbrugger of Vienna in 1761. However, the value of percussion was not fully appreciated until the year 1808, when the French physician Corvisart, physician to Napoleon I., revived the subject and further elaborated the technic of percussion. During the same epoch Piorry of France introduced topographical percussion of the thoracic viscera, and Skoda of Vienna published his studies upon the probable genesis of hyperresonance and tympany in connection with pulmonary relaxation and compression. The technic of percussion and the attributes and mode of production of the various percussion findings were further elucidated by the epoch-making researches of such investigators as Wintrich, Traube, Biermer, Gerhardt, and Weil. Indeed, so thorough were the studies undertaken by these latter investigators, that modern investigation has accomplished little in the way of additions to the physical laws which were enunciated by these authors in relation to the generation of the various characteristics of the percussion sounds.

Percussion is employed in the study of disease of the respiratory organs for the purpose of eliciting sounds which are normal to the pulmonary parenchyma and sounds which only arise in the presence of diseased states of these organs; to determine the situation of the borders of the intrathoracic organs; and also to note the degree of resistance offered to the percussion stroke by the tissues under examination.

Percussion, as commonly practiced, consists in striking the surface of the area under examination with a view primarily to eliciting sound, and secondarily for the determination of the degree of resistance offered to the percussion blow. During this maneuver the percussion blow may be directed with the finger or with a specially devised percussion hammer, and either directly upon the part under examination, or upon an intervening medium, usually a finger of the opposite hand of the examiner, or in other instances plates of metal, glass, ivory, or hard rubber. The instrument with which the blow is struck, finger or hammer, is termed the *plexor*; the intervening medium, finger or plate, is termed the *pleximeter*. When an intervening medium or pleximeter is employed, the percussion is termed *mediate percussion;* whereas when no such intervening instrument is present, but the blow is delivered directly upon the part which is under examination, the percussion is termed *immediate percussion.*

Immediate Percussion.—In the practice of immediate or direct percussion the pleximeter is dispensed with and the blow is struck directly upon the chest wall. The finger-tips or the palm of the hand may be employed, the former in the percussion of the clavicles to determine the presence of consolidation of the apices of the lungs, and the latter in lightly slapping the two halves of the thorax in the effort to demonstrate the presence of dullness over a relatively large area, usually at the bases posteriorly.

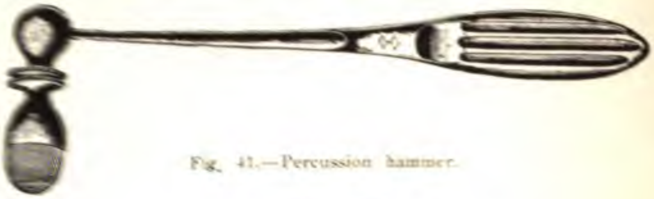


Fig. 41.—Percussion hammer.

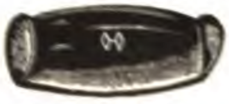


Fig. 42.—Hard rubber pleximeter.

Mediate Percussion.—This, the most frequently employed mode of percussion, is practiced by two methods; namely, by *instrumental percussion*, and by *finger percussion*. Each method has its inherent advantages; but it is advisable for the student to perfect himself in one method and to discard the other.

In instrumental percussion the blow is directed upon a pleximeter of glass, ivory, rubber, or metal by means of a special percussion hammer. It is essential that the pleximeter shall be sufficiently narrow to fit into the intercostal spaces without overlying the adjacent ribs, and it is necessary that the plexor be equipped with a head of soft rubber in order to avoid the production of adventitious sound during the delivery of the percussion blow.

In the practice of finger percussion a finger of the left hand of the examiner is applied firmly and evenly with the palmar aspect down upon the area under examination while the blow is delivered by one or more fingers of the right hand, the plexor finger or fingers being flexed as nearly as possible at a right angle. The stroke should preferably be directed upon the base of the nail or upon the second phalanx of the middle finger of the pleximeter hand.

To obtain satisfactory data by finger percussion it is essential that the pleximeter finger be applied firmly and evenly upon the part; that the percussion stroke be delivered quickly and in a vertical direction; that the plexor finger be raised immediately and not be permitted to remain in contact with the pleximeter; that the percussion blow be delivered entirely by a wrist movement, the forearm not participating; and that the successive blows be delivered with uniform intensity.

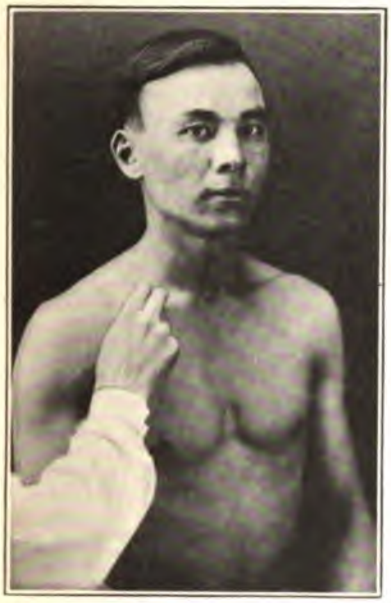


Fig. 43.—Immediate percussion of clavicle.

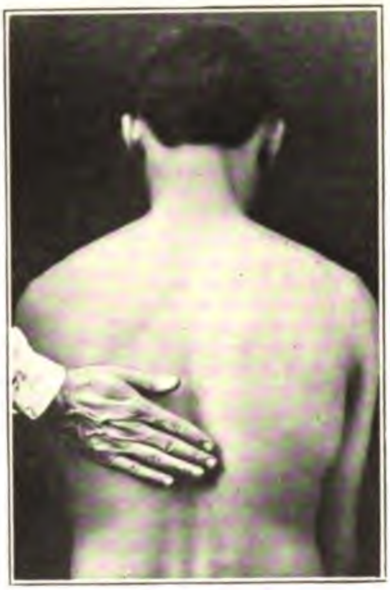


Fig. 44.—Immediate percussion of pulmonary bases.

Finger percussion is preferable to instrumental percussion for the reason that the pleximeter finger becomes a sensitive palpating medium, appreciating minor variations in the degree of resistance which the chest wall offers to the percussion stroke. Moreover, the finger may be more firmly applied to the area under investigation, excluding the possible intervention of air between chest wall and pleximeter.

In the practice of mediate percussion only a few strokes should be employed in a given region. As much information is to be obtained by four or five strokes properly directed as by a greater number, which tend to impair the nicety of the auditory appreciation of the tones which are elicited. In comparing the quality of the sound elicited upon the two sides of the chest, exactly corresponding points should be selected upon either side; and the percussion blows should be delivered with uniform force in each instance.

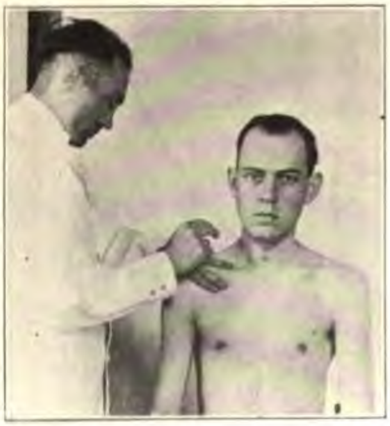


Fig. 45.—Percussion of pulmonary apices.

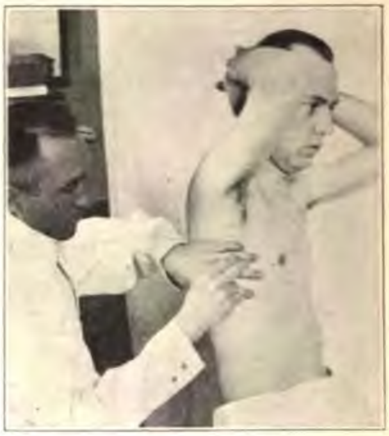


Fig. 46.—Percussion of lateral thoracic region.

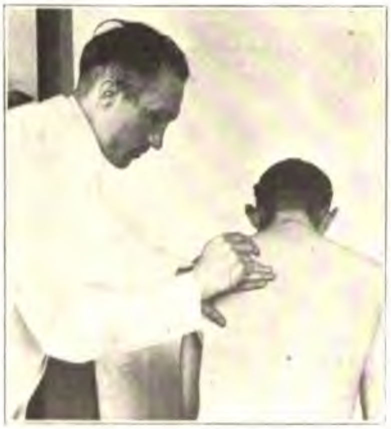


Fig. 47.—Percussion of posterior thorax.

Percussion is practiced with most satisfactory results with the patient in the sitting or standing posture. During percussion of the anterior surface of the thorax, as well as in percussion of the supraclavicular regions, the hands should hang naturally at the sides, the head should be held symmetrically in the median plane of the body, and the entire attitude of the patient should be natural and unconstrained. In percussion of the supraclavicular and infraclavicular regions, the examiner, assuming a position at the side of the patient should apply the pleximeter fingers firmly and evenly to the integument in these regions, the index and middle fingers occupying the infraclavicular region and the ring finger occupying the supraclavicular region. During percussion of the lateral regions of the thorax a good exposure is obtained by directing the patient to clasp the hands behind the head, the arms meanwhile being drawn backward. In percussion of the posterior thoracic wall the patient should bend the trunk forward, meanwhile crossing the arms upon the chest, and approximating the elbows. In this attitude the scapulæ are widely separated and are closely applied to the bony thorax. When it is necessary to practice percussion with the patient in the recumbent posture the examiner should bear in mind the adventitious impairment of resonance in the dependent portion of the thorax; and the patient must be turned a sufficient number of times to insure the percussion of all regions of the thorax in the most favorable posture obtainable.

PALPATORY PERCUSSION

In the practice of this method of percussion the primary object is the determination of the degree of resistance afforded by the underlying structures rather than the production of sound. Palpatory percussion is employed in both direct and indirect methods. In the former the chest wall is struck with the pads of the finger-tips with a pushing movement, the sensitive finger-tips noting the degree of resistance to the blow. Indirect or mediate palpatory percussion is practiced by striking the pleximeter finger with a tardy, pushing movement rather than with the sharp, quick stroke of routine percussion, and allowing the plexor to remain for an instant upon the pleximeter.

Palpatory percussion is of service in determining the presence of fluid in the pleural cavity and in outlining the borders of a hypertrophied heart or a large pericardial effusion by the gradations which are encountered in the resistance to the percussion blow

AUSCULTATORY PERCUSSION

This method of percussion, which combines auscultation with mediate or immediate percussion, is employed for the purpose of outlining the borders of solid organs, consolidations, neoplasms, and collections of fluid. In the practice of auscultatory percussion the chest-piece of the stethoscope is applied over the organ, consolidation, or structure which is to be outlined, and is retained in position by the patient or an assistant while the examiner, after first percussing near the bell of the instrument and fixing in his mind the quality of the note which is elicited, proceeds to percuss toward the instrument from the several directions upon the surface of the thorax. In the performance of this maneuver mediate percussion may be employed or direct palpatory percussion, or immediate percussion in the form of rapid finger flicking. Instead of employing percussion, the same result is obtained if the vibrating tuning-fork is drawn over the integument of the thorax toward the bell of the instrument; or, in the absence of this instrument, by drawing the rubber eraser of an ordinary pencil over the skin.

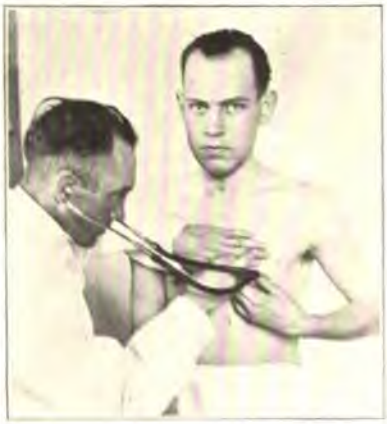


Fig. 48.—Auscultatory percussion.

In auscultatory percussion of solid structures which are covered by pulmonary tissue the note is observed to undergo two successive alterations: a primary impairment of resonance, which is followed secondarily by frank dullness or flatness. The several points at which the percussion note is observed to change may be marked upon the skin with a dermographic pencil; and, when connected by a line drawn through each of them, will indicate the borders of the organ or structure under examination. Most frequently employed in outlining the areas of relative and absolute cardiac dullness, auscultatory percussion is also useful in differentiating by tonal variations between the dullness of the liver and that due to pneumonic consolidation of the right base or pleural effusion of the right side. Similarly, it is an invaluable aid in differentiating consolidations of the left lung from the dullness of the heart.

RESPIRATORY PERCUSSION

This mode of percussion consists in percussion of the two sides of the thorax during quiet respiration and again during forced inspiration with the object of determining minor variations in the pulmonary resonance upon the two sides of the thorax. The method is particularly useful in the detection of minor degrees of consolidation. In incipient phthisis upon consecutive percussion during quiet and during forced inspiration, the dullness upon the consolidated side remains unaltered, while the sound side exhibits an increase of resonance.

SUPERFICIAL AND DEEP PERCUSSION

The terms superficial and deep as applied to percussion have special reference to the force of the percussion blow, and each method has its indications in the examination of the thoracic viscera. In practicing superficial percussion the thorax is tapped quickly and lightly, whereas in deep percussion the blow is delivered with sufficient force to set up vibrations in the deeper structures. In the examination of persons with thin chest walls as well as in the examination of children, superficial percussion is to be employed. It is also to be preferred in the delimitation of consolidations which are situated immediately beneath the chest wall, in order to obviate the establishment of confusing vibrations in the deeper structures.

In the examination of patients with thick chest walls, on the contrary, and in the elicitation of dullness of deep consolidations which are covered by normal pulmonary tissue, forcible or deep percussion is to be employed. This method of examination is useful in eliciting the dullness of central pneumonia, deeply situated aortic aneurysm, and in estimating the upper limit of hepatic dullness.

ATTRIBUTES OF THE PERCUSSION SOUND

The sound which is elicited upon percussion of the thorax possesses certain inherent attributes or properties; namely, *quality, pitch, intensity* or *volume,* and *duration.*

Quality.—Quality is the property or attribute by which a given sound is distinguished from a sound of different origin. It is by the quality of the sounds that the sound which is produced upon striking a piece of iron is distinguished from that which is produced upon striking a piece of wood, and by which the sound which is produced upon percussing over the normal lung is differentiated from that which is elicited upon percussing over a solid organ. It is their quality which gives to the various sounds which are elicited upon percussion their individuality and much of their localizing value.

Pitch.—The pitch of a sound is determined by the rapidity of the vibrations by which the sound is produced. When the surface of the thorax overlying the lung is percussed, the air content of the innumerable pulmonary alveoli is thrown into vibration. Pitch may be high or low, depending as it does upon the rapidity of these vibrations, rapid vibrations producing a note of high pitch, while slow vibrations produce one of low pitch. The rate of the vibrations is in turn influenced by the size of the cavity containing the air and by the force of the percussing blow, being more rapid in small cavities and with forcible percussion blows.

Intensity.—Intensity, or volume, has reference to the loudness of the sound, this in turn depending upon the amplitude of the vibrations which are generated, the force of the percussion blow, the thickness of the chest wall, and the amount of air in the area under examination. With heavy percussion over an area of lung containing an excess of air and with a thin chest wall, an intense sound is produced; while a similar stroke over a region containing little air, overlaid by thick chest walls, produces a sound of minor intensity.

Duration.—The duration of length of the percussion sound possesses less of diagnostic significance than do the other attributes. In general, it may be stated that the clearer the note and the higher the pitch, the shorter the duration; the duller the note and the lower the pitch, the longer the duration.

DEGREE OF RESISTANCE

Aside from eliciting sound, percussion is employed to determine the degree of resistance as appreciated by the pleximeter. In many instances the degree of resistance encountered affords a valuable information as does the sound which is elicited; and i cases where for any reason the sounds produced are not typical it may be the sole guide of the examiner. Increased resistanc to the percussion blow as appreciated by the pleximeter indicate a decrease in the air content of the part and a correspondin increase in solid structure or the presence of fluid. A hig pitched note with well marked sense of resistance indicates tha the air content is small while the proportion of solid material i correspondingly in excess of the normal amount.

NORMAL PERCUSSION SOUNDS

Pulmonary Resonance

The sound which is elicited upon percussion of the surfac of the thorax which overlies normal crepitant pulmonary tissu is termed *pulmonary resonance*, or *normal vesicular resonanc* When the thoracic surface is percussed, the aerial content of th pulmonary infundibula and alveoli which are situated within th range of the shock of the percussion blow is caused to vibrate, wit the consequent production of sound. The quality of this sound i distinctive, and is only afforded by percussion of normal pulmonar tissue containing its normal quota of air separated by innumerabl interalveolar septa. The intensity and pitch of the sound depen upon the amplitude and rate of the vibrations which are generate in the aerial content of the closed spaces, by the distance of thes air-containing spaces from the thoracic surface, and by the thickness of the chest walls. With strong percussion, vibrations o greater amplitude are generated with the production of a mor intense sound than that which is produced by feeble percussio Similarly percussion of the thorax in the infraclavicular and axi lary regions, situations in which the pulmonary tissue is closel apposed to the thoracic walls is attended by a more intense soun than is percussion of the region of the shoulder girdle where th lung is separated by voluminous musculature and bony structur from the surface of the thorax. Similarly in the thin thorax of th female subject and the child the resonant note of percussion of th normal lung possesses greater intensity than in the correspondin region in the normal adult male subject.

The normal thorax presents certain *regional variations* in pu monary resonance with which it is essential that the examin be familiar. Pulmonary resonance of maximum purity is obtain upon percussion of the first two intercostal spaces in the infraclavicular region for the reason, as Seitz has shown, of the greater width of the intercostal spaces, and the relative thinness of the thoracic wall in this region. In these interspaces, however, the resonance exhibits minor variations in its intensity. It attains its maximum intensity in the midclavicular line, from which point it diminishes in intensity as one percusses outward toward the shoulder where the great pectoral muscle becomes more voluminous, and inward toward the sternum, in which situation the anterior border of the lung becomes progressively thinner, and presents a less voluminous pulmonary parenchyma in the sphere of the shock of the percussion blow. In right-handed subjects the resonance is usually slightly less intense upon the right side of the thorax in the first two interspaces, owing to the more excessive development of the pectoral muscles upon this side. It is, moreover, frequently noted that the resonance which is elicited in the first intercostal space is slightly less intense than that which is produced upon percussion of the second interspace, a fact which is explained by the fact that the first intercostal space is more deeply situated than is the second owing to the backward course of the first rib beneath the clavicle, and by the fact that in the act of mediate percussion the pleximeter is forced in the majority of cases to rest partially upon these bones.

During percussion of the first two intercostal spaces, careful comparison of the intensity of the resonance should be made upon the two sides of the thorax. In percussion of the regions of the anterior thoracic surface below the second interspace such comparison is useless on account of the influence which the projection of the heart toward the left has upon the intensity of the two sounds.

In the third and fourth intercostal spaces the pulmonary resonance is less pure than in the first two spaces, owing to the extensive distribution of the great pectoral muscle in these regions, to the thickness of the panniculus adiposus of the mammary gland, and, upon the left side, the projection of the heart to the left of the sternal border. Seitz holds that the more pronounced narrowing of the intercostal spaces as one descends upon the anterior thoracic wall plays an important part in the progressive enfeeblement of pulmonary resonance which is encountered

In the fifth intercostal spaces pulmonary resonance is variable upon the two sides of the thorax. Upon the left side of the thorax in the male subject the intensity of the resonance is moderately impaired by the presence of the mammary gland, and markedly

impaired by the same gland in the female subject. Mor
internal to the midclavicular line upon this side of the thorax
area of relative cardiac dullness, corresponding to the regi
which the heart is invested by the anterior pulmonary b
which becomes progressively less voluminous, there is marke
pairment of pulmonary resonance. Similarly, upon the righ
of the thorax in the fifth interspace the area of relative h
dullness is encountered in which the upper diaphragmatic s
is clothed by the thin lower border of the lung, yielding a di
tion of resonance which is soon to terminate in frank flatness
the area is attained in which the liver is directly apposed
anterior thoracic wall. In the right fifth intercostal spa
areas of relative cardiac and relative hepatic dullness m
almost a right angle, forming an angle of pulmonary reso
immediately to the right of the sternum in this interspace, *Eb*
cardiohepatic angle.

In the sixth right intercostal space pulmonary resonance e
an abrupt transition into flatness, indicating in this situati
superior limit of the area of hepatic flatness, in which the li
in immediate apposition, beneath the diaphragm with the an
thoracic wall.

In all regions of the thorax percussion over the ribs and
structures yields pulmonary resonance of less purity tha
which is obtained upon percussion of the intercostal spaces
deed, as one percusses inward in the superior intercostal
toward the sternum, as this bone is encountered, the *osteal*
nance afforded by percussion of this bone blends with the pulm
resonance of the intercostal spaces.

In the supraclavicular regions pulmonary resonance is en
ered which becomes progressively more intense as one per
inward toward the median line, to finally give place in this lo
to the tympany of the trachea. The progressive increase in i
ity of pulmonary resonance under these circumstances should
all cases attributed to its real cause, the proximity of the t
to the pulmonary apices in the median line of the body. Of
different significance is hyperresonance encountered in
regions, when this is encountered throughout the region a
tends well outward toward the distal extremity of the clavicl

Percussion of the clavicle yields resonance of impaired int
which contrasts markedly with the purity of the pulmona
nance in the infraclavicular region.

further impairs the purity of the resonance as do also irregularities due to former fractures or osseous disease.

Percussion of the sternal region, overlying the sternum and ensiform cartilage, yields three distinct variations in pulmonary resonance. Aside from the influence which the osteal resonance of the bone exerts upon the pulmonary resonance, there are distinct variations in the quality and intensity of the pulmonary resonance as a result of the influence upon the latter of the anatomic structures with which the sternum is in relation in different portions of its distribution. Percussion of the manubrium sterni and the portion of the gladiolus above the level of the fourth costal cartilage yields upon forcible percussion resonance blended with the tympany of the subjacent trachea and primary bronchi. Below this level percussion of the gladiolus yields rather marked impairment of resonance, corresponding to the area of relative cardiac dullness in which the heart is covered by the thin borders of the lungs. Percussion of the ensiform cartilage yields frank dullness or flatness, on account of its apposition with the left lobe of the liver. However, in the presence of extreme distention of the stomach with gas, this flatness may be replaced with gastric tympany, which has nothing in common with the quality of pulmonary resonance.

Upon the lateral walls of the thorax pulmonary resonance is somewhat less intense than upon the anterior wall; but it is decidedly more intense than is the case upon the posterior wall of the thorax. In this regard, the three aspects of the thorax present the same relative variations in regard to the intensity of pulmonary resonance as was observed in the same regions in the study of vocal fremitus.

However, whereas in the case of vocal fremitus the tactile vibrations are observed with slightly exaggerated intensity upon the right lateral wall of the thorax, in the case of pulmonary resonance the reverse obtains; pulmonary resonance is slightly diminished upon the right lateral thorax, a diminution which is caused by the same factors which make for increased vocal fremitus in this area.

In the axillary region pulmonary resonance is slightly less intense than in the infraaxillary region; but in the upper axillary region the note is slightly tympanitic on account of the closer proximity to the chest wall in this region of the larger bronchi. Upon percussion of the lateral thoracic wall the resonance is observed to augment in intensity as one descends until the seventh intercostal space is reached. At this level upon the right side of the thorax pulmonary resonance is impaired by the advent of the area of relative hepatic dullness; while at the same level upon the left side of the thorax, it is replaced by the tympanitic sound generated in the stomach and colon.

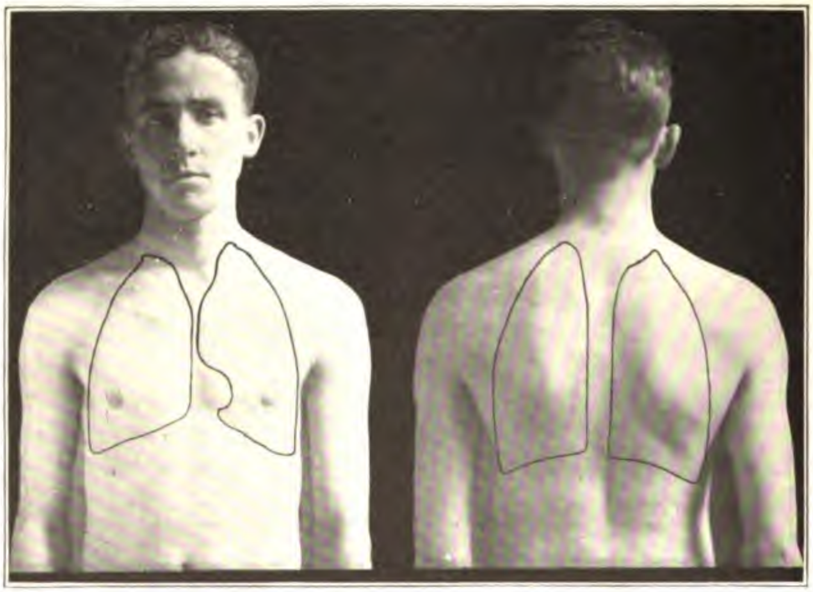
In every portion of the posterior surface of the thorax pulmonary resonance is less intense than upon the anterior and lateral regions of the thorax. Upon the posterior thoracic wall resonance of greatest purity is obtained upon percussion of the pulmonary bases in the infrascapular region. Next in order of purity comes the resonance in the inferior half of the interscapular region, and this is followed by the superior half of the same region. In the suprascapular and scapular regions, overlying the bony scapulæ with their thick investment of musculature, pulmonary resonance is less intense than in any other portion of the posterior thoracic surface. In this region the minimum intensity is encountered along the course of the spine of the scapula.

The Normal Limits of Pulmonary Resonance.—The normal limits of pulmonary resonance correspond accurately to the anatomical site of the borders of the lungs. The superior limit extends into the base of the neck from one to one and one-half inches above the clavicles, extending a short distance higher over the right than over the left apex. Anteriorly the limits of resonance lie behind the sternum in the upper portion of the thorax, the osteal resonance of this bone interfering greatly in the delimitation of the anterior pulmonary borders in this region. At the level of the fourth intercostal space, however, the anterior limit of resonance is displaced to the left by the interposition of the heart between the left lung and the chest wall, in the areas of relative and absolute cardiac dullness. The inferior limits of normal pulmonary resonance are encountered at the level of the sixth rib in the midclavicular line, the eighth rib in the midaxillary line, and the tenth rib in the scapular line.

Variations in the Limits of Pulmonary Resonance.—In bronchopulmonary disease any lesion which increases the expansion of the lung in any direction results clinically in a corresponding increase in the area of pulmonary resonance; and any lesion which decreases the extent of the lung in any direction produces a corresponding limitation of the resonance to the extent of the lesion. It follows that in disease of the lung or pleura there may be a general increase or decrease of resonance at all of the pulmonary borders; that the variation may involve two corresponding borders; or that only a single area of the lung may present an abnormal variation in the extent of pulmonary resonance.

A general increase of pulmonary resonance in all directions, all of the borders of the lungs occupying a position beyond their normal limits, is characteristic of hypertrophic emphysema and is noted during the paroxysm of bronchial asthma. A similar general increase is frequently noted in dyspneic patients with uncompensated heart disease, and occasionally in subjects of chronic bronchitis.

A general decrease of pulmonary resonance, due to general retraction of the pulmonary borders, occurs with atrophic emphysema, in which the lungs are symmetrically shrunken and atrophic.



A. *B.*

Fig. 49.—Limitation of pulmonary resonance at apices. *A.* Anterior view. *B.* Posterior view.

Extension of resonance at the apices occurs in connection with the paroxysms of bronchial asthma and pertussis as a result of the acute vesicular emphysema which attends these attacks. When hypertrophic emphysema involves the apices extensively, there is extension of the limits of resonance in the supraclavicular regions.

Decreased resonance at the apices, associated with dullness of the percussion note and depression of the supraclavicular fossæ, is a valuable sign of chronic ulcerative phthisis. Retraction of the limit of resonance in this direction also occurs with fibroid phthisi apical pneumonia, pulmonary collapse from bronchial obstructio and as a result of traction of adhesions in chronic adhesive pleuris The apical limitation of resonance in phthisis is commonly b lateral, whereas that of apical pneumonia and chronic adhesi pleurisy commonly involves one apex.

Extension of resonance of the anterior borders of the lungs s that they encroach upon or obscure the area of cardiac dullne occurs in established hypertrophic emphysema. During the tra sient acute vesicular emphysema of the paroxysm of bronchi asthma and of pertussis the anterior border of the left lung e croaches upon the area of cardiac dullness.

Decreased resonance of the anterior border of one or both lun is indicative of fibroid retraction of the lung in chronic interstiti pneumonia or fibroid phthisis or of displacement of the pulmonar border by a hypertrophied heart or by pericardial or pleur effusion. In the case of fibroid retraction of the left lung t cardiac impulse is diffuse, occupying a wide area in the thir fourth, and fifth interspaces; whereas in the presence of pleural c pericardial effusion the impulse is displaced or is invisible.

Increased resonance of the lower border of the lungs is part an parcel of the general extension of resonance accompanying hype trophic emphysema or bronchial asthma. It may also occur wit fibrinous bronchitis and uncompensated heart disease.

Decreased resonance of the lower borders of one or both lun points to fibroid retraction of the lung due to chronic interstiti pneumonia or fibroid phthisis; or to elevation of the diaphragm d to paralysis of that muscle or to the subphrenic pressure of ascite abdominal tumor, hepatic, or splenic enlargement. In atelecta and in pleurisy with effusion the lower border of the lung is lik wise elevated with diminution in the area of pulmonary resonan in this direction.

THE RESPIRATORY EXCURSION OF THE LUNG

In estimating the respiratory excursion of the lungs, the low limit of pulmonary resonance should be defined by percussion i the midclavicular, midaxillary, and scapular lines during qui and during forced inspiration, and the difference noted. Usuall measuring approximately one inch, the respiratory excursion very much reduced in subjects of hypertrophic emphysema an during asthmatic attacks, as well as in cases of limited di phragmatic excursion occurring as a result of increased subphrenic pressure or painful thoracic affections.

ABNORMAL PERCUSSION SOUNDS

Impaired Resonance.—The percussion note exhibits slight impairment of the purity of vesicular resonance, not amounting to dullness, which is the next gradation, when there is only a moderate increase in the solid over the normal crepitant structure of the lung. Impaired resonance is elicited particularly in the early stages of phthisis at the apices of the lungs. Impairment of resonance is also a sign of moderate pleural thickening and incipient consolidation from pneumonia or other cause. It is the first step toward dullness, but is not so pronounced in its change of quality.

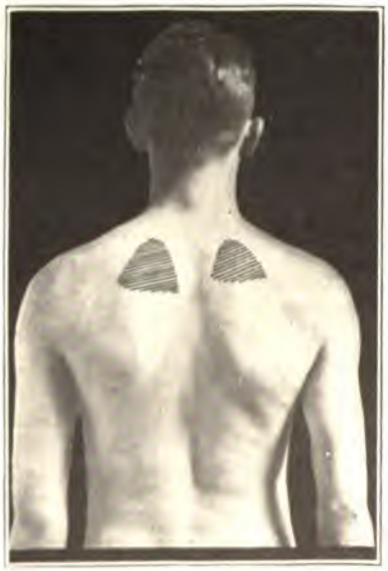


Fig. 50-*A*.—Areas of dullness in apical pulmonary tuberculosis.

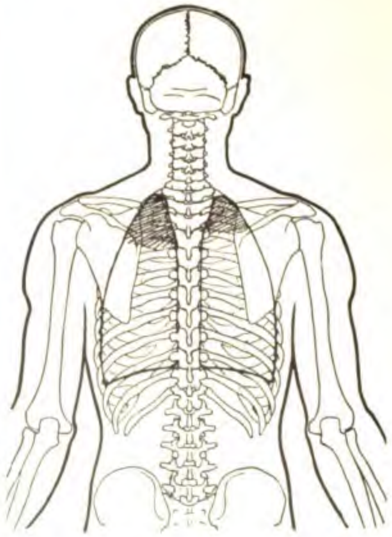


Fig. 50-*B*.—Areas of dullness in apical pulmonary tuberculosis.

The note which is elicited upon percussion over airless structures which are separated from the chest wall by the intervention of normally crepitant lung is termed *relative dullness*. This note is elicited in percussion of the areas of relative cardiac and relative hepatic dullness, in which these organs are overlapped by the borders of the lungs.

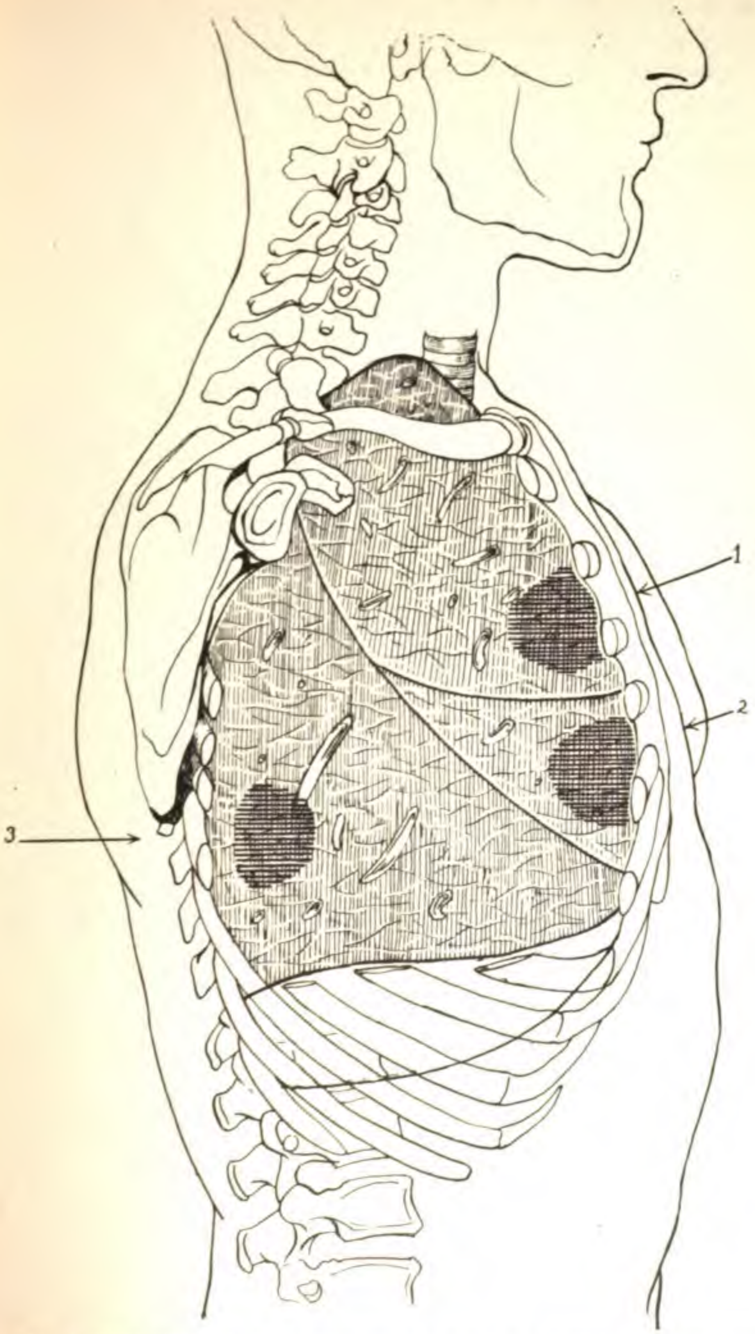


Fig. 51.—Physical causes of change in percussion note. *1-2*, dullness upon percussion; *3*, deep dullness masked by intervening lung.

Dullness.—As in the case of impaired resonance, dullness indicates a decrease in the air-content of the part and a corresponding increase in the solid elements in the area under examination; but the note is more materially altered than it is in impairment of

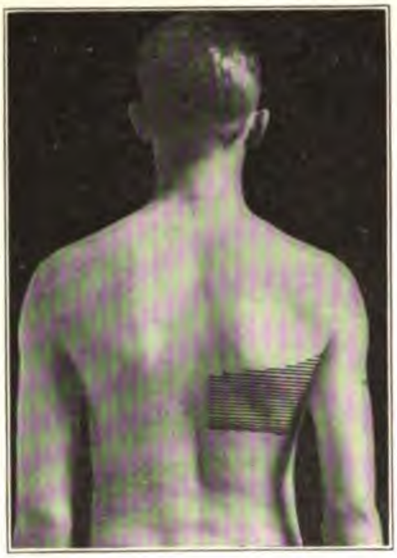


Fig. 52-*A*.—Area of dullness in moderate pleural effusion.

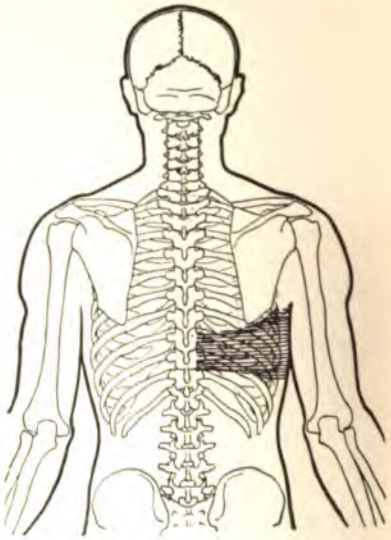
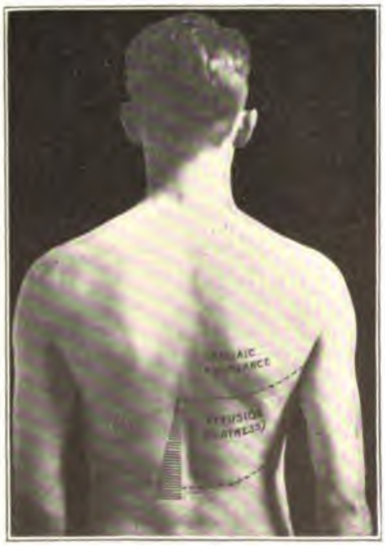
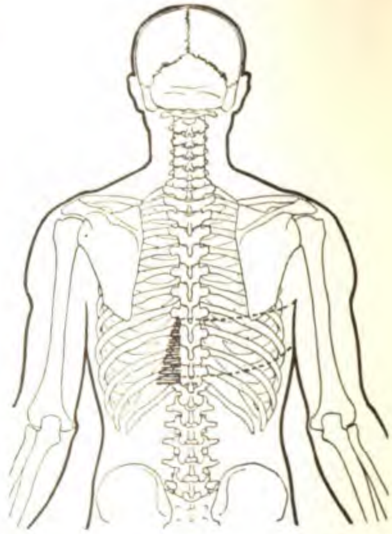


Fig. 52-*B*.—Area of dullness in moderate pleural effusion.





resonance. A dull note is elicited upon percussion in the presence of the consolidations of pneumonia and phthisis, infiltration of the lung with edema and hypostatic congestion, in carcinomatous infiltration, in the presence of considerable pleural thickening, in the area of a lung which is compressed by a tumor, in atelectasis which is extensive in distribution, and in pulmonary syphilis and in hemorrhagic infarction of the lung.

Dullness localized to a special region is encountered in pleurisy with effusion, owing to compression or condensation of the mediastinal structures and their deflection toward the side of the thorax opposite to the effusion. Thus, in pleurisy with effusion there is frequently a triangular area of paravertebral dullness opposite the side of the effusion, at the level of the twelfth dorsal spine, constituting *Grocco's sign* of this disease.

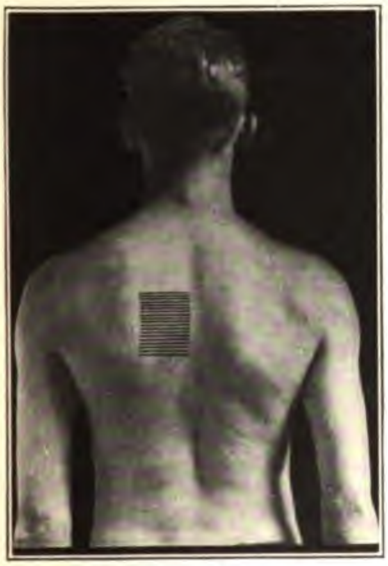


Fig. 54-*A*.—Dullness of aortic aneurysm.

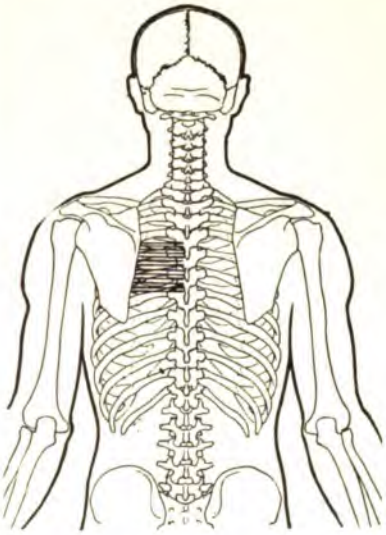


Fig. 54-*B*.—Dullness of aortic aneurysm.

Dullness which is circumscribed to the left interscapular region is noted in connection with aneurysm of the thoracic aorta. Dullness in this region may, however, signify enlargement of the bronchial glands or mediastinal tumor. Apical dullness, when associated with normal pulmonary resonance over other portions of the lung, points to incipient phthisis; but similar dulling of the note in this region is present in apical pneumonia and in pleural thickening.

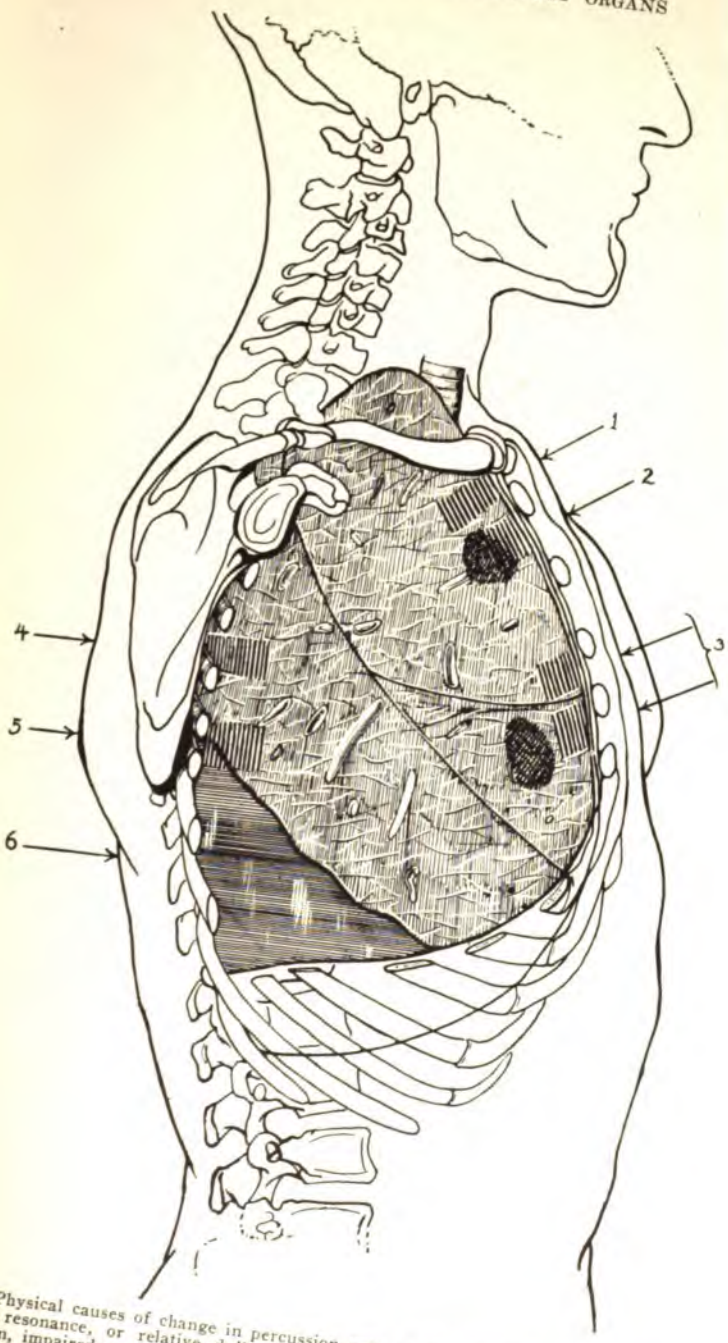
Flatness.—Flatness, a percussion note which is entirely devoid of resonance, is indicative of entire absence of air from the area percussed. It is elicited upon percussion over a consolidated lobe in fibrinous pneumonia, over a pleural effusion, a tumor of the lung or pleura, a greatly hypertrophied heart, a cirrhosed lung, hydrothorax, or a pulmonary cavity which is filled with fluid. A deeply seated consolidation, which is overlaid by normal lung, frequently fails to give dullness or flatness owing to compensatory emphysema of the intervening lung and requires deep percussion to elicit dullness.

Flatness of one base posteriorly, associated with skodiac resonance of the apex of the corresponding lung, frequently accompanied by Grocco's sign in the opposite paravertebral region, is indicative of serofibrinous pleurisy or empyema. Flatness in the same area, shifting with change of posture is very suggestive of hydropneumothorax. Flatness anteriorly, obscuring the normal gastric tympany of Traube's semilunar space, occurs with left-sided pleural effusion, while flatness in Ebstein's cardiohepatic angle accompanies right-sided effusion.

Hyperresonance.—Hyperresonance, an increase in normal vesicular resonance, is characterized by an abnormal clearness of the percussion sound, as a result of an increase of the air content of the area which is percussed. Hyperresonance may be bilateral, unilateral, or localized to a limited area of the chest. When bilateral hyperresonance is elicited, it usually indicates hypertrophic emphysema; when unilateral, compensatory emphysema is the usual underlying cause; while localized hyperresonance is elicited over a small portion of a lung which is the site of vicarious distention to compensate for an adjacent focus of compression, collapse, or consolidation. Such a localized area of hyperresonance is often indicative of a deeply seated patch of consolidation, which requires deep percussion to indicate its presence.

TYMPANY

Tympany represents the acme of hyperresonance, percussion of the thorax yielding in this instance a tympanitic or drum-like sound. Its prototype in the human economy is produced by percussion over the stomach when this viscus is moderately distended with gas and by percussion over the larynx and trachea. Tympany is encountered upon percussion over pulmonary cavities which contain air, whether these possess a patent bronchial



55.—Physical causes of change in percussion note. 1, normal vesicular resonance;
aired resonance, or relative dullness; normal vesicular resonance
cussion, impaired resonance upon deep percussion; 4,
ss; 6, flatness of fluid origin.

communication or are closed, and upon percussion of pulmonary tissue which is in a state of relaxation or compression.

The intensity of the tympanitic note depends upon the amplitude of the vibrations which are generated within the area percussed, upon the distance of the area in question from the thoracic walls, and upon the thickness of these walls. The intensity of the sound is greater in the case of large superficial cavities than in the case of cavities of smaller size owing to the greater mass of air which is set in vibration and the readiness with which the vibrations are transmitted to the exterior of the thorax.

The pitch of the tympany depends upon the size of the cavity; upon the presence or absence of a patent bronchial communication; upon the diameter of the bronchial communication when such is present; and upon the tension of the walls of the cavity. In interpreting variations in the pitch of the tympanitic sound in any case the student should bear in mind the physical principles governing the pitch of the tympanitic sound as generated by percussion over pulmonary excavations; namely, that the pitch of the tympany is in inverse proportion to the length of the column of air which is set in vibration upon percussion of the cavity, and in direct proportion to the diameter of the bronchial communication, if there be one; that the shorter the air column which is set in vibration, or in other words, the smaller the dimensions of the cavity, the higher is the pitch of the tympany; that the greater the diameter of the bronchial communication, the higher is the pitch, and the smaller the transverse diameter f the bronchial communication, the lower is the pitch; that of wo *closed* cavities the tympany will be of higher pitch in the one hich shall have the lesser vertical diameter, and vice versa; and hat percussion of a large closed cavity may yield a tympanitic ote of higher pitch than a smaller cavity which has a bronchial mmunication of small diameter, the lowering of the pitch in the ter instance being determined by the narrow orifice of the nchial communication. From this last consideration it follows before basing conclusions as to the probable dimensions of a monary cavity upon the pitch of the tympany, it is necessary to blish whether the cavity is closed or open by the changes of d of Wintrich and Friedreich, to be described in a subsequent graph.

influence of the length of the air column upon the pitch of mpanitic percussion note is readily demon sing a pleximeter successively above the orifices of glass cylind which are filled with varying quantities of water, or upon percuss above the mouth of a glass cylinder while an assistant slowly po water into the cylinder along its walls. Under these circumstan it is observed that as the water mounts in the cylinder and as column of air becomes progressively smaller, the percussion n becomes progressively higher in pitch.

The influence of the diameter of the orifice of the bronch communication upon the pitch of the percussion note can be dem strated in an experimental manner by percussing above gl

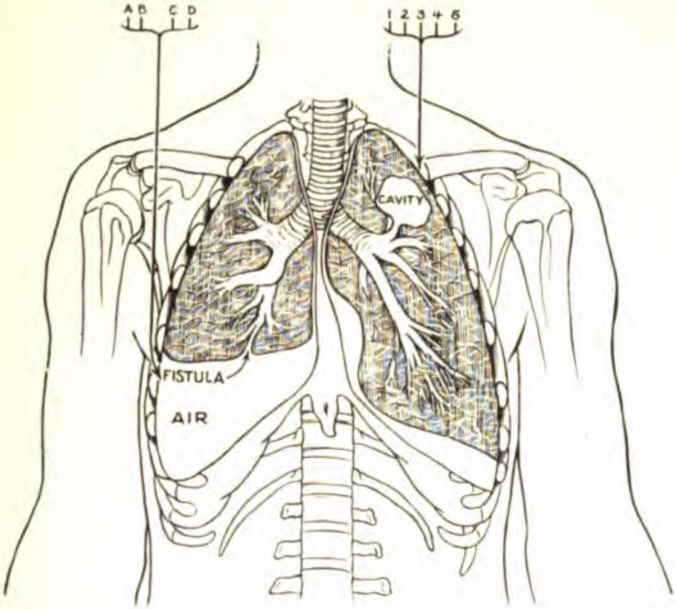


Fig. 56.—Physical basis of pathologic physical signs upon percussion and auscult of the thorax.

A, cracked-pot sound; *B*, Biermer's phenomenon; *C*, pectoriloquy; *D*, lung-fi sound; *1*, cracked-pot sound; *2*, Wintrich's change of sound; *3*, Friedreich's chang sound; *4*, bronchial or amphoric respiration; *5*, whispering pectoriloquy.

cylinders which are covered with pieces of cardboard in which perforations of variable diameter, or by percussing above a sin cylinder, first with the mouth of the cylinder uncovered, and s cessively as it is covered with pieces of cardboard with perforati of progressively descending diameter. Under these circumstan it will be observed that the percussion tympany is of highest pi in the case of the uncovered cylinder, and that its pitch beco progressively lower as the orifice of the cylinder becomes sma

The mechanism of generation of the

and its quality when generated are in very intimate relationship the one with the other. Upon percussion of a pulmonary cavity with smooth walls vibrations are created in the air of varying amplitude. In the case of cavities with smooth walls and with free bronchial communication the state of affairs is in some degree analogous to that which obtains in the pipes of an organ or other wind instrument in which the quality and pitch of the notes depend upon variations in the length of the vibrations which are produced. Like the pipe of the organ, a pulmonary cavity with smooth walls and free bronchial communication acts as a resonator for certain of the vibrations which are created in the air content upon percussion. The quality of the tympany which is elicited depends upon the rate of vibration of the fundamental vibrations, which are the slowest vibrations which the chamber is capable of responding to, and the rapidity of these vibrations is in direct relation to the length of the column of air contained in the cavity, or, in other words, to the length of the cavity. It is thus that is explained the change of sound of Wintrich, which will be discussed in a subsequent paragraph.

It is essential that a cavity possess smooth walls in order to yield a tympanitic sound upon percussion; and when the walls of a cavity become coated with fibrinous exudate or tissue debris, the tympanitic character of the sound elicited upon percussion is lost. Wintrich found in his experiments that upon percussion above the orifice of a cylinder sunk in soft, dry snow that he obtained tympany, whereas upon percussion above the ragged depression left after removal of the cylinder that tympanism was absent. He also demonstrated the absence of tympany upon percussing above vessels of leather, felt, and other materials of irregular surface.

A pulmonary cavity must attain certain dimensions in order to yield a tympanitic percussion sound. Wintrich places the minimum diameter at six centimeters; while Skoda failed to obtain tympany in cavities approximately the size of the fist.

While all pulmonary cavities which contain air and possess a patent bronchial communication present the physical requirements for the production of tympany upon percussion, it frequently happens that cavities are too deeply seated in the lung to yield tympany upon percussion. It is only when such a cavity is situated in the periphery of the lung that tympany is to be elicited, and even in these cases cavitation is frequently not detected by the inexperienced examiner

The tympanitic percussion sound is capable of exhibiting f changes of sound under appropriate conditions; namely, the *cha of sound of Wintrich*, the *interrupted change of sound of Wintr* the *respiratory change of sound of Friedreich*, and the *chang sound of Gerhardt*.

WINTRICH'S CHANGE OF SOUND

Wintrich demonstrated that upon percussion of a superfi pulmonary cavity with patent bronchial communication tympany is of higher pitch with the patient's mouth open an lower pitch when the lips are tightly closed during the percuss The accepted physical basis for this change in pitch is, in br that the buccal cavity, which acts as a resonating chamber the vibrations which are conducted to it from the pulmon cavity by way of the respiratory passages, is tuned for differ vibrations accordingly as the lips are opened or as they closed; that is to say, that with the lips closed the buccal ca acts as a resonator for the waves of longer amplitude and lo rate of vibration, whereas with the lips parted the buccal ca is adapted best to the waves of less extensive amplitude and m rapid vibration, which it magnifies to the exclusion of the slo vibrations of greater amplitude.

In the elicitation of the change of sound of Wintrich the pat should occupy the seated position, as Baümler has shown during examination in the dorsal decubitus the root of the ton often tends to close more or less completely the orifice of larynx and to prevent the change of pitch of the percussion so upon opening the lips. Also the change of sound should be elic during the same phase of the respiratory cycle, as the chin the glottis is subject to changes in diameter during the two piratory acts. Moreover, the act of deglutition, by occlu the laryngeal orifice effectually masks the change of sound.

WINTRICH'S INTERRUPTED CHANGE OF SOUND

When a pulmonary cavity contains fluid which is freely mo and shifts its position readily with change in the posture of patient, alternately occluding and leaving unoccluded the b chial communication of the cavity, there is frequently witne the alternate presence and absence of Wintrich's change of so upon changes in the attitude of the patient. When the pos of the patient is such that the bronchial communication is situated above the level of the fluid, Wintrich's change of sound is demonstrable upon percussion; whereas with the shifting of the fluid and occlusion of the bronchus with change of posture the change of sound is abolished. As a result of the entrance of air beneath the surface of the fluid which is contained within the cavity, gurgling râles are not infrequently audible upon the change of the patient's posture.

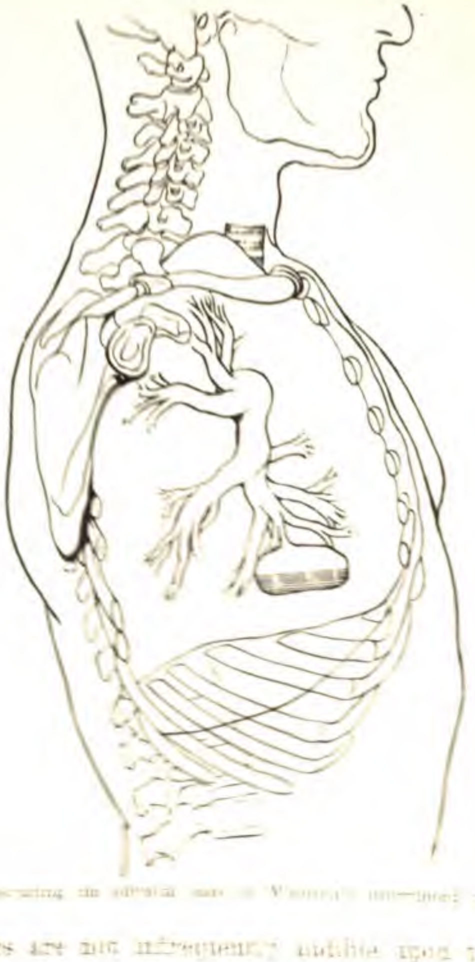


Fig. 37.—Illustrating the physical basis of Wintrich's interrupted change of sound.

When this sign can be elicited, it is of service in determining whether the bronchial communication of the cavity is situated upon its base, or upon the anterior, posterior or lateral wall of the cavity. If the bronchus communicates with the base of the cavity, with the patient in the upright attitude the fluid occupies the base of the cavity and effectually prevents the change of sound of Wint rich. Upon placing the patient in the dorsal decubitus, the mobil fluid occupies the posterior portion of the cavity and the sign o Wintrich reappears. If the bronchial orifice is situated on th posterior wall of the cavity, the opposite condition of affairs ob tains, and the change of note is demonstrable in the erect positio and is abolished in the dorsal decubitus. If, on the other hand, th change of sound is demonstrable in both the erect posture and in th dorsal decubitus, but is abolished upon placing the patient in th genupectoral position, the bronchial orifice is evidently situate upon the anterior wall of the cavity. If, finally, the change o sound persists in all three of the above-named attitudes, the

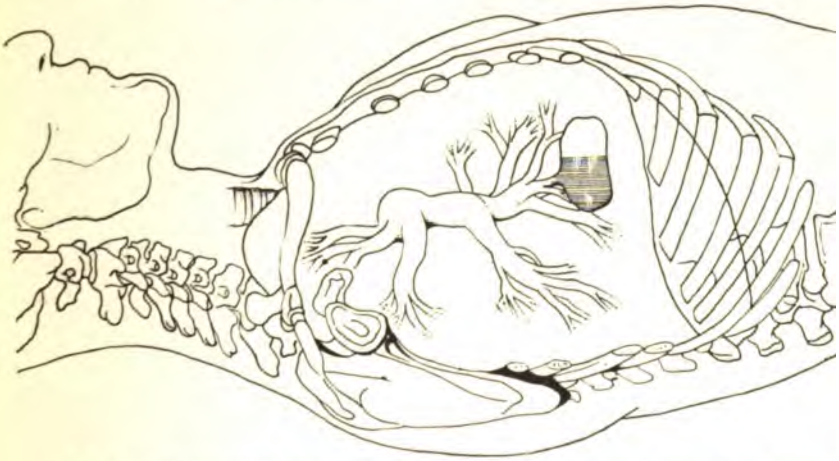


Fig. 57-*B*.—Illustrating the physical basis of Wintrich's interrupted change of sound.

the bronchial orifice is situated upon one of the lateral walls o the cavity, and the change of note is obliterated upon placing th patient upon the side of the orifice.

FRIEDREICH'S RESPIRATORY CHANGE OF SOUND

Friedreich noted that the tympany which is elicited upon pe cussion of a superficial pulmonary cavity with patent bronchia communication possesses a higher pitch during inspiration tha during expiration. This change of pitch is explained upon th basis of the increased diameter of the chink of the glottis durin inspiration, the glottic slit in this instance, in continuous com munication through the bronchial system with the pulmonar vity, acting as the true orifice of the cavity and its breadt determining the pitch of the percussion tympany during the two phases of respiration. Moreover, the increased tension of the walls of the cavity during inspiration causes a slight increase in the pitch of the percussion note, which at the same time loses a portion of its tympanitic quality. Of definite diagnostic value when present, this sign is rather difficult to elicit in most cases.

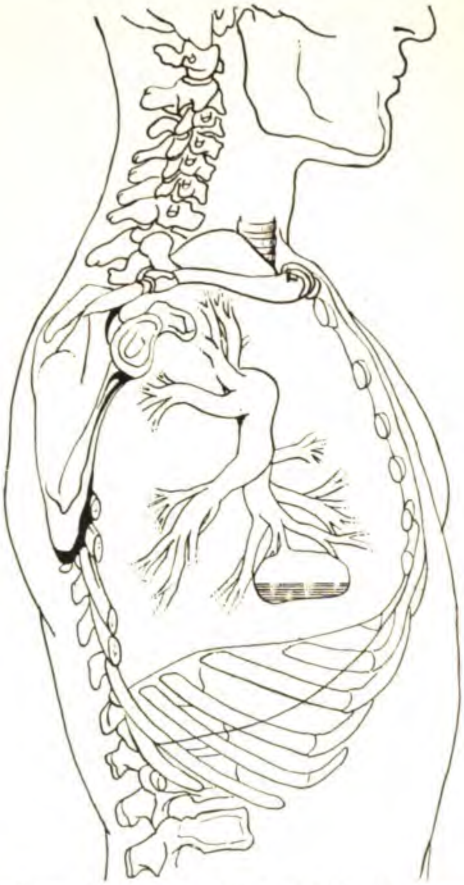


Fig. 58-*A*.—Illustrating the physical basis of Gerhardt's change of sound.

GERHARDT'S CHANGE OF SOUND

When a superficial pulmonary cavity is partially filled with mobile fluid, and when its vertical and horizontal diameters are unequal, there is noted a definite change in the pitch of the percussion note with changes in the posture of the patient. When the subject is so placed that the greater diameter of the cavity is horizontal, the tympany elicited upon percussion is of distinctly lower pitch than it is when the greater diameter of the cavity is vertical, as the result of variations in the tension of the walls of the cavity. Thus, it is observed that this sign may be utilized in the case of closed cavities in the estimation of the direction of the greater diameter of the cavity; but, as in the case of open cavities similar changes of note are observed with changes of posture, it is essential in the first place to determine through the signs which have been described whether or not the cavity is closed.

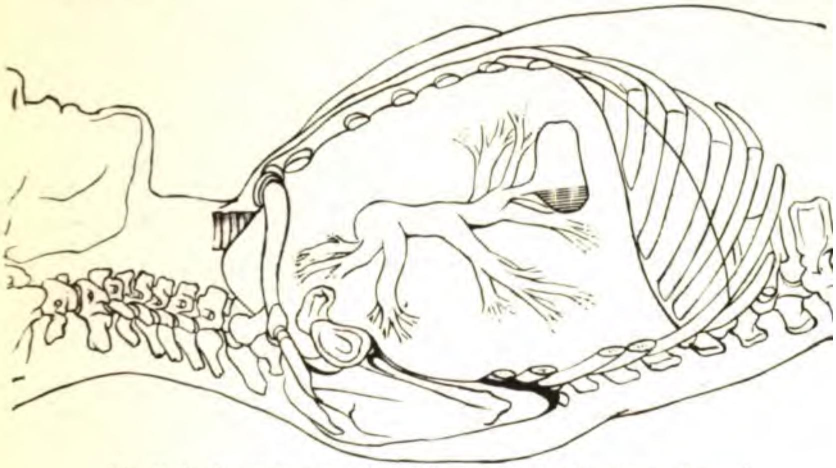


Fig. 58-*B*.—Illustrating the physical basis of Gerhardt's change of sound.

BIERMER'S PHENOMENON

Biermer noted a change of sound in the presence of hydropneumothorax analogous to the change of sound of Gerhardt; namely, that the tympanitic note elicited upon percussion over a hydropneumothorax was of lower pitch when the patient was placed in the recumbent posture, and changed to a note of higher pitch when the patient assumed the sitting posture. The variation in this instance is attributable to the change in the relative diameters of the air-containing cavity, as a result of gravitation of the fluid to the most dependent portion of the sac.

SKODAIC RESONANCE

Skodaic resonance is a tympanitic sound which is elicited upon percussion of a portion of the lung which is in a state of relaxation or of compression. This variety of tympany may be elicited in the presence of disease of the bronchi, alveolar disease, or in disease of organs adjacent to the lungs, as the pleura, pericardium, heart, or the abdominal viscera. Skodaic resonance is differentiated from tympany due to pulmonary excavation by the fact that the pitch of the tympanic sound is uniform and is not altered upon opening or closing the mouth or upon changes of posture.

In respect to the relative frequency of the etiologic factors which are concerned in the generation of Skodaic resonance, pulmonary compression stands first and foremost, and is followed by alveolar disease, and by bronchial affections.

In the presence of extensive pleural effusion the lung is elevated and is crowded into the superior portion of the pleural cavity, leading to compression and relaxation of the alveoli of the upper lobes. Hence, in this disease the examiner frequently encounters Skodaic resonance upon percussion of the thorax above the level of the fluid in these conditions. In the case of extensive pericardial effusion pressure is exerted upon the mediastinal surface of the upper lobe of the left lung, leading to compression and relaxation of this portion of the organ and the production of Skodaic resonance upon percussion of the first and second left intercostal spaces. Upon the posterior surface of the thorax, on the contrary, below the angle of the left scapula, the examiner commonly encounters dullness upon percussion, associated with bronchovesicular or bronchial breathing and increased vocal fremitus, due to a compression of the inferior lobe of the left lung to such a degree that the air is in the main forced from the alveoli, constituting Bamberger's sign of this disease. Immense cardiac hypertrophy is not infrequently attended by Skodaic resonance, which is distributed along the superior and left lateral boundaries of the area of relative cardiac dullness. Occasionally, though by no means frequently, Skodaic resonance is elicited over the superior portion of the thorax in the presence of upward displacement of the diaphragm by excessive intraabdominal pressure due to ascites, tympanites, or large abdominal tumor.

Skodaic resonance is present in the case of certain affections of the pulmonary alveoli, when the latter contain simultaneously fluid and semisolid materials. These conditions are met during the first and the third stages of lobar pneumonia, during catarrhal pneumonia, during pulmonary edema, and in pulmonary infarction of extensive distribution. The presence of dullness over both bases posteriorly with Skodaic resonance over the superior portions of the thorax anteriorly is very suggestive of catarrhal pneumonia. Hutinel and Paisseau hold that Skodaic resonance in the infraclavicular regions is the first intimation of the presence of infantile catarrhal pneumonia, making its advent before the development of impaired resonance is manifested over the bases posteriorly.

When, in the development of acquired atelectasis due to bronchial obstruction, the obstruction becomes complete, the air which is contained in the alveoli distal to the bronchial occlusion is gradually absorbed, and with the consequent relaxation of the pulmonary tissues, Skodaic resonance makes its appearance.

WILLIAMS' TRACHEAL TONE

Upon percussion of the apices of the lungs in the supraclavicular and infraclavicular regions, in the presence of apical consolidation the percussion sound frequently has imparted to it the tympany of the adjacent trachea, constituting the tracheal tone of Williams. As the fundamental cause of the sound is the vibration which is generated in the aerial content of the trachea, the tympany of Williams is subject to the variation in pitch of Wintrich upon opening and closing the mouth. In the elicitation of the note, forcible percussion is employed over the supraclavicular and infraclavicular regions.

AMPHORIC RESONANCE

Amphoric resonance is a tympanitic note of clanging, echoing, metallic quality, and of decidedly longer duration than pure tympany. A similar resounding note is elicited upon striking the side of a barrel which is empty or partially filled with water, or upon speaking in a loud voice in an empty chamber with high ceiling or in a vaulted cellar.

Amphoric resonance is generated in percussion of superficial pulmonary cavities of irregular configuration, and with smooth, tense walls, which are free from secretion. The sound is elicited over both open and closed cavities, and the pitch of the note varies with the vertical diameter of the cavity. Wintrich explained the generation of the amphoric sound on the assumption of the creation during percussion of vibrations in the air content of varying amplitude, the more rapid overtones dying away more quickly than does the fundamental tone of greater amplitude, producing an echoing sound of considerable duration.

The intensity of the amphoric percussion sound is influenced by the thickness of the walls of the cavity, by the distance of the cavity from the thoracic wall, by the thickness of the thoracic wall, and by the force of the percussion blow. In the case of open cavities, the intensity is increased when the mouth is opened.

THE CRACKED-POT SOUND (BRUIT DE POT FÊLÉ; MONEY-CHINK RESONANCE)

The cracked-pot sound is a variety of tympany in which the percussion note bears some resemblance to the sound which is produced upon tapping the side of a cracked metal jar, or to the muffled chink of coins, whence the name money-chink resonance. Neither of the above comparisons gives a true conception of the quality of the sound, which is hissing rather than metallic in quality; and its quality is more aptly imitated by the sound which ensues upon suddenly striking the clasped palms upon the knee with the sudden escape of the air confined between the palms of the lung and that the thoracic parietes be sufficiently thin and elastic to permit the expulsion of the air from the cavity during the delivery of the percussion blow. Grancher has noted the disappearance of the cracked-pot sound over superficial cavities after a few percussion blows and its reappearance following several deep inspirations. He assumes that the air is totally expelled from the cavity during the primary percussion, and that the sound only again becomes evident upon replacement of the aerial content of the cavity during deep inspiration. In the case of a cavity containing fluid or viscid secretion, abolition of the sound may be due to bronchial occlusion.

In the presence of apical pneumonia, apical consolidation of chronic phthisis, or in compression of the superior lobe of the lung by an immense pleural effusion it is occasionally possible to obtain a cracked-pot sound upon percussion of the anterior thoracic wall in the first and second intercostal spaces. In this case the cracked-pot sound is likely to be attended by the tracheal tone of Williams.

During the first and third stages of lobar pneumonia the cracked-pot sound is occasionally demonstrable in the infraclavicular and mammary region upon the side of the disease. Cockle has observed the sign in simple catarrhal bronchitis in children; and Rollet has encountered it in bronchopneumonia.

In the case of open pneumothorax, whether the fistulous opening be external upon the surface of the thorax, or internally as a result of rupture of a tuberculous cavity of the lung, the cracked-pot sound is frequently to be elicited. Nothnagel, Oppolzer, Frerichs, and Eichhorst have encountered the sound in association with this disease.

When the cracked-pot sound is encountered at the apices, it is plainly a question of pulmonary compression or of pulmonary cavity with bronchial communication, which is probably tuberculous in origin. In cases of tuberculous origin Loeb holds that the sound is most frequently encountered over the right apex.

Bronchiectatic dilatations of the bronchi furnish all of the physical requirements for the generation of the cracked-pot sound, the chink of the glottis in this instance playing the rôle of the constricted orifice through which the air is expelled during percussion. However, bronchiectases are usually too deeply seated in the pulmonary parenchyma to be materially influenced he force of the percussion blow, and, moreover, bronchiectasis disease which is commonly encountered.

GAIRDNER'S COIN TEST (BELL TYMPANY; ANVIL TEST)

In cases of pneumothorax, when the stethoscope is applied to the base of the thorax posteriorly while an assistant percusses the anterior surface of the thorax with two coins employed as pleximeter and plexor, respectively, an echoing, metallic sound is appreciated through the stethoscope, possessing a ringing quality analogous to the distant ring of a hammer upon an anvil.

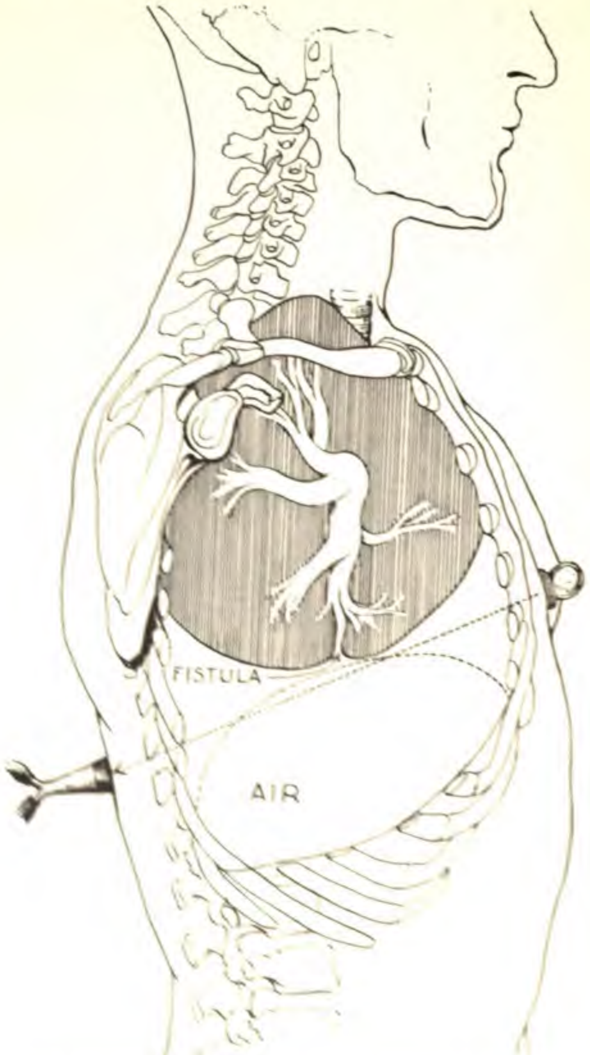


Fig. 59.—Illustrating bell tympany in Gairdner's coin test.

CHAPTER V

AUSCULTATION

Object and Technic.—Auscultation is the act of listening to sounds which are generated within the thorax or abdomen with the unaided ear or with a specially devised instrument, the stethoscope. When the unaided ear is employed, the procedure is termed *immediate auscultation* in contradistinction to instrumental auscultation, which is termed *mediate auscultation*. Auscultation, which is the most important single method of physical examination, is employed in the study of the lungs, heart, and digestive organs, and in eliciting certain vascular phenomena.

Stethoscopes are monaural and binaural, as they are equipped with one or two earpieces, respectively. The monaural instrument is seldom employed by American clinicians, having yielded place to several types of binaural stethoscope. The modern binaural stethoscope consists of a bell or chestpiece of hard rubber or metal, which is connected with the earpieces by rubber tubing, the principle involved being the clear transmission of sound from the subject to the ear of the examiner and the exclusion as far as possible of all extraneous noises. In the Bowles stethoscope the bell is replaced by a chestpiece containing a vibrating diaphragm. This type of instrument is very useful in the examination of bedridden patients and during hasty examination of female subjects, when the chestpiece is readily slipped beneath the clothing. Stethophones have been employed in the practice of auscultation, the object of the instruments being to magnify the intensity of the sounds which are elicited; but their use should be guarded, as they are poor conductors of high-pitched sounds, and from a clinical standpoint the clearness and quality of the sounds are of greater importance than is an artificial magnification of the intensity of the sounds elicited. In the selection of a stethoscope, whatever type is adopted, care should be exercised to secure an instrument whose bell and tubing are sufficiently heavy to exclude as far as possible extraneous noises and whose earpieces fit snugly in the ears of the examiner.

Immediate Auscultation.—In the practice of immediate auscultation the ear of the examiner is applied directly to the part under examination, only a thin garment or a towel intervenin between the ear and the chest wall. The position of the patie should conform to that which obtains during mediate auscult tion.

Mediate Auscultation.—In the practice of mediate auscultati the bell of the stethoscope is applied firmly and evenly to t chest wall, but without the exertion of undue pressure. The b of the instrument is retained in position by grasping it near t

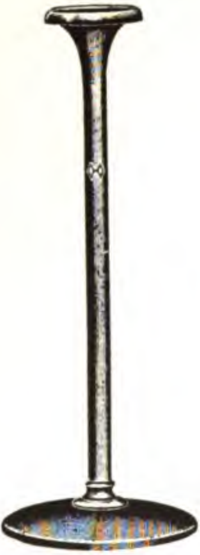


Fig. 60.—Hawksley's monaural stethoscope.

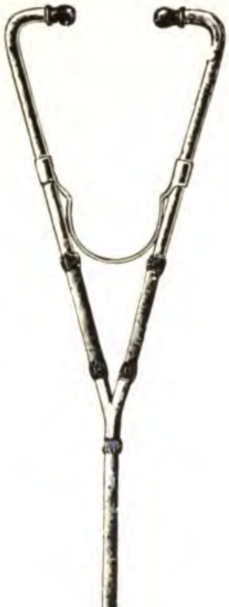


Fig. 61.—Bowles stethoscope.

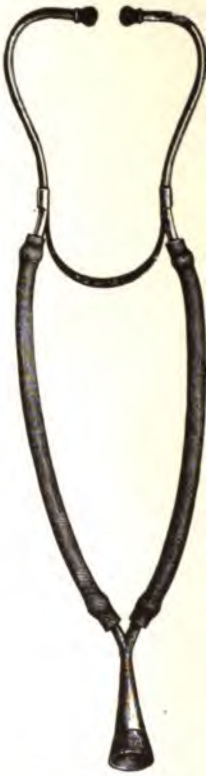


Fig. 62.—Binaural stethoscope.

base with the index finger and the thumb. No article of weari apparel should be permitted to intervene between the bell of t instrument and the surface of the thorax.

During auscultation of the respiratory organs the examin should note the character of the sounds which are generat during quiet, moderately deep, and forced respiration, being e on the alert for any deviation from the normal sounds

Auscultation is practiced by preference with the patient in the sitting posture. During auscultation of the anterior surface of the thorax the arms of the patient should hang naturally at the sides, and his position should be natural and free from undue muscular tension. In auscultation of the axillary and infra-axillary regions the patient should raise the arms laterally only so far as is necessary to ensure a free access to this portion of the thorax. During auscultation of the posterior aspect of the thorax the patient should incline the trunk forward and clasp the arms across the chest to ensure a wide separation of the scapulæ.

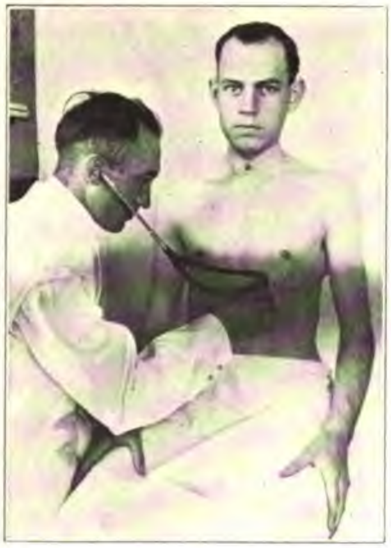


Fig. 63.—Auscultation of thorax.

Auscultation of the thorax should be practiced methodically, the examiner progressing downward from the apices to the bases of the lungs. In each instance the sound which is elicited upon one side should be compared with the sounds elicited over the corresponding area upon the opposite side of the thorax. Wandering and hasty auscultation of the thorax, without comparison of the sounds upon the two sides, is a productive source of error in physical examinations.

The ear of the examiner should be trained to disregard all extraneous noises, such as the friction produced by the rubbing together of the tubes of the instrument, the contact of the hand with the instrument, and crepitation due to contact of the be of the stethoscope with a hairy chest wall. This last annoying feature may be eliminated by moistening the bell of the instrument prior to its application to a hairy chest wall. The beginner in auscultation usually experiences some difficulty in separating the pulmonary and the cardiac sounds; but concentration and practice will enable him to disregard the one while studying the other.

NORMAL RESPIRATORY SOUNDS

Upon auscultation of the various regions of the thorax of the normal subject three types of respiratory murmur are encountered; namely, *bronchial breathing*, *vesicular breathing*, and *bronchovesicular breathing*.

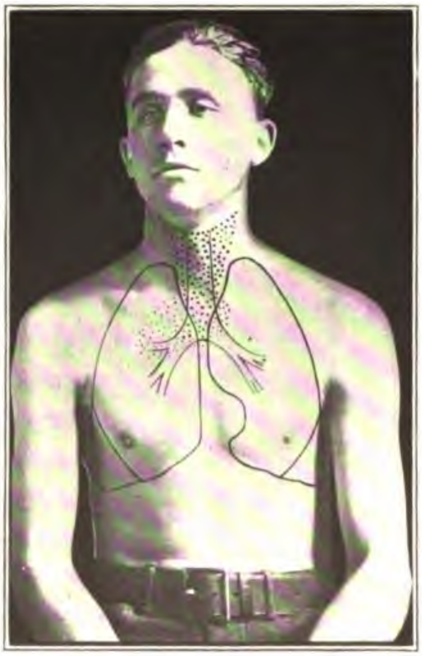


Fig. 64.—Normal distribution of bronchial and bronchovesicular breathing. Anterior thoracic surface.

Bronchial Breathing.—Bronchial breathing is a blowing, tubular sound, the inspiratory and expiratory phases of which are as a rule of equal duration, though occasionally expiration is slightly prolonged. The inspiratory and expiratory phases of the murmur are separated by a distinct interval; and the intensity and pitch of the two phases are different, for physical reasons which wi be detailed in succeeding paragraphs.

Bronchial breathing in its maximum purity and intensity i elicited upon auscultation of the larynx and trachea, over the sit of production of the aerial whorls which call the sounds int being. The sound is also commonly audible in the interscapula regions in the area situated between the seventh cervical an fourth dorsal vertebra, and over the manubrium sterni anteriorl; the latter two levels corresponding to the level of the trache bifurcation. These are the regions of the thorax in which bror chial breath sounds are encountered during tranquil respiratior

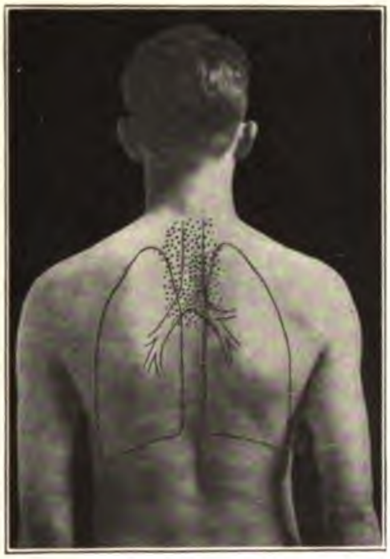


Fig. 65.—Normal distribution of bronchial and bronchovesicular breathing. Posteri thoracic surface.

In the presence of extreme grades of dyspnea, with marked ir crease in the force and depth of the respirations, it is not ur common for bronchial breath sounds to become audible over th entire thoracic surface.

When during inspiration the inspired air passes through th orifice of the glottic slit and enters the wider cavity of the laryn beyond, aerial whorls are generated in the air content of th cavity, whorls which are conducted downward into the trache and larger bronchi in the form of the inspiratory phase of bror l breathing. Similarly, when during expiration the expire ısses through the chink of the glottis into the broad pharyngeal cavity beyond, similar whorls are induced with the production of the expiratory phase of bronchial breathing.

As the genesis of the sound in each instance lies in the passage of air through the glottic slit, it is easy to comprehend the influence of the diameter of this opening upon the intensity and the pitch of the two phases of bronchial respiration. The physical laws governing aerial currents teach us that the intensity of the sound engendered by such currents varies in direct proportion with the degree of stenosis or narrowing of the orifice. Hence, the expiratory phase of bronchial respiration, corresponding to the narrowing of the glottis at this time, is more intense than the inspiratory phase of bronchial breathing. Similarly, since according to the physical laws governing the pitch of sounds generated in this manner the pitch is in direct proportion to the size of the opening, one readily understands that the inspiratory phase of bronchial breathing, corresponding to the widening of the chink of the glottis during inspiration, is of distinctly lower pitch than is the expiratory phase.

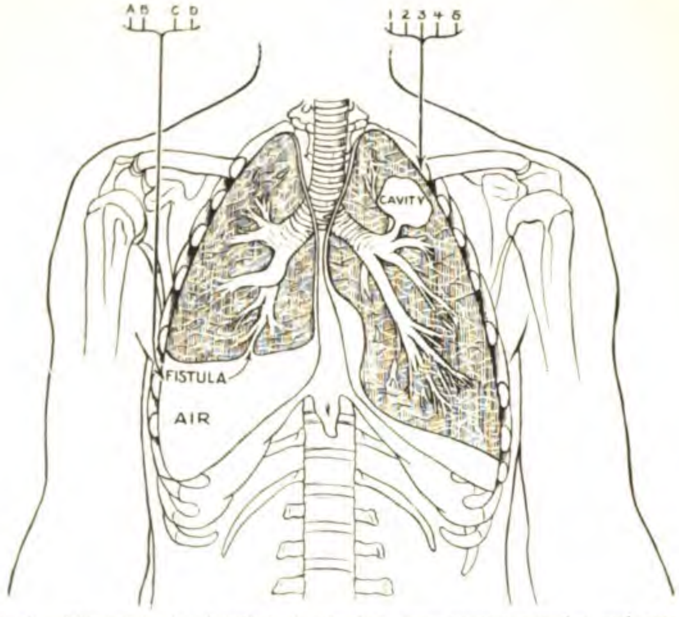


Fig. 66.—Physical basis of pathologic physical signs upon percussion and auscultation of the thorax.

A, cracked-pot sound; *B*, Biermer's phenomenon; *C*, pectoriloquy; *D*, lung-fistula sound; *1*, cracked-pot sound; *2*, Wintrich's change of sound; *3*, Friedreich's change of sound; *4*, bronchial or amphoric respiration; *5*, whispering pectoriloquy.

The tendency for these vibrations to be conducted downward throughout the bronchial system and outward to the thoracic w by way principally of the aerial content of these tubes a secondarily by the walls of the tubes themselves, is combated the great mass of aerated pulmonary parenchyma which int venes between the principal bronchi and the thoracic walls. the presence of extensive consolidation of the pulmonary alve however, and in the presence of extensive cavitation of the lu with free communication with the bronchial system, the bronch sounds generated in the larynx are conducted to the thoracic w with an intensity and clearness which justifies the descript term of "tubular breathing."

Vesicular Breathing.—Upon auscultation of the infraclavicu and mammary regions anteriorly, of the axillary and infraax lary regions laterally, and of the infrascapular regions pos riorly, regions of the thoracic surface which overlie portions the lungs which are comparatively remote from the princi bronchial tubes, the respiratory sounds possess a soft, bree sound of low pitch, which is termed vesicular or alveolar brea ing. The murmur consists of two phases, inspiratory and expi tory, which are separated by a short interval. The inspirat murmur is maintained approximately three times as long as the expiratory murmur. The quality of vesicular breathing been compared to the sound produced by the soft rustling of leaves of a tree when agitated by a gentle breeze. The quality the sound can also be rather closely simulated by approaching lips nearly in contact and inspiring strongly.

The physical genesis of the vesicular murmur has not as been accounted for in an adequate manner. Laennec erroneou attributed the sound to the rubbing of the inspired and expi air against the walls of the bronchi and pulmonary alveoli, explanation which rests upon no adequate physical basis. Blak ton held that during inspiration the involuntary muscular fibe disposed in a circular manner around the walls of the smal bronchioles, contracted with the consequent production of part stenosis of these tubules with the generation of the vesicu murmur, a theory which is not substantiated entirely from eit the physiologic or physical point of view. Leaning would h us believe that the murmur is a purely muscular sound, genera by contraction and relaxation of the smooth muscle bands of bronchioles during inspiration and expiration. Gerhardt attr uted the vesicular murmur to vibrations of the alveolar w during inspiration. At the present time th planation of the sound, it would seem, is that of Baas and Penzoldt, who state that the vesicular murmur is merely bronchial breathing softened in its transmission from the larynx along the aerial column contained in the trachea and bronchi, mingled with the soft crepitation of innumerable pulmonary alveoli during their inflation and subsequent collapse.

Whatever may be the precise genesis of the vesicular breath sound, clinical experience has taught us to construe its·presence to mean that in any region of the thorax where it is encountered, the pulmonary alveoli and bronchioles are permeable to air. It does not follow from this, however, that its presence assures a normal condition of these structures. In the small, disseminated patches of consolidation attending miliary tuberculosis of the lungs and lobular pneumonia it is possible for the intervening aerated areas of the lung to suppress any enfeeblement of the murmur which might arise as the result of the exclusion of the air from the minute and scattered areas of alveolar consolidation.

The intensity of the vesicular murmur is dependent upon the force of the respiratory efforts and the thickness of the thoracic walls in the normal subject. The influence of the depth of the respiratory act upon the murmur is well illustrated upon auscultation of the thorax during Cheyne-Stokes respiration, when the intensity of the vesicular murmur will exhibit all gradations from accentuation of the murmur at the height of the dyspneic period to its total abolition during the period of comparative apnea. In children and in the female subject, owing to the thinness and elasticity of the thoracic parietes the vesicular murmur is more intense than is the case in the adult male subject. The age has a direct bearing upon the intensity of vesicular breathing. Up to the twelfth year of life, vesicular respiration is more intense than it is subsequent to that age; while in the aged subject there is a progressive diminution in the vesicular element of the respiratory sounds, which come to approach the quality of bronchial breathing or bronchovesicular breathing.

The pitch of vesicular breathing varies with the age and sex of the subject. It is uniformly of higher pitch in the infant and young child than it is in the adult subject; and it is of higher pitch in the female than in the male subject. The variation in the pitch of the murmur is in each of these instances caused by the smaller diameter of the glottic slit in the first class of subjects. Again, in the aged subject, without regard to sex, there is an increase in the pitch of vesicular breathing, the result in this instance of senile rarefaction of the pulmonary tissues.

Bronchovesicular Breathing.—This type of respiration, as its name implies, combines the qualities of bronchial and of vesicular breathing. It is audible over regions of the normal thorax in which the larger bronchi are in fairly close proximity to the thoracic wall, but are nevertheless covered by aerated pulmonary tissue. Thus, bronchovesicular breathing is normally audible over the lower portion of the manubrium sterni and adjacent to its right lateral border anteriorly, and in the interscapular regions posteriorly at the level of the fourth dorsal vertebra.

ABNORMAL RESPIRATORY SOUNDS

In the presence of disease of the respiratory organs, various modifications of the normal respiratory sounds are encountered. The alteration may consist of the presence of bronchial or bronchovesicular breathing in a region of the thorax where it is not normally present; of the assumption by bronchial breath sounds of certain cavernous or amphoric qualities which are not normally attributable to them; of variations in the intensity of the vesicular murmur in regions of the thorax where it is normally present; or of variations in the frequency or rhythm of the respiratory sounds.

Bronchial Breathing.—When bronchial breath sounds are detected in a region of the thorax where they are not normally audible, it most frequently points to consolidation of the lung, the solidification conducting the murmur from the large bronchi to the surface of the thorax. Other factors which act similarly comprise pulmonary compression and collapse, hemorrhagic infarction, cirrhosis of the lung, enlarged bronchial glands, a tumor overlying a large bronchus, or a pulmonary cavity situated near the chest wall with a free bronchial communication.

Bronchial breathing which is referable to a cavity with patent bronchial communication frequently has a peculiar hollow quality engrafted upon it to which the term *cavernous breathing* is applied. In this subtype of bronchial breathing expiration is frequently of lower pitch than is inspiration. Similarly, a cavity with patent bronchial communication or a pneumothorax with an open bronchial fistula often gives rise to bronchial breathing of rather musical quality, closely simulating the sound which is generated by gently blowing across the mouth of an empty bottle,

oric breathing.

Vesicular Breathing.—In disease of the respiratory organs the intensity and rhythm of the normal vesicular murmur is frequently so altered as to possess definite diagnostic significance.

Diminution in the intensity or entire abolition of the vesicular murmur is normal in aged subjects and in subjects with very thick chest walls. The murmur is also diminished in the presence of painful diseases of the chest wall such as incipient pleurisy and pleurodynia, which cause the patient to inhibit the respiratory excursions of the chest. A similar diminution occurs with moderate pleural thickening, edema of the lung, the early stage of lobar pneumonia, and in the presence of a closed pneumothorax.

Abolition of the vesicular murmur is noted over a region of the thorax in which the main bronchus is occluded, over a large pleural effusion, and over a pulmonary cavity which is filled with fluid.

Increased Intensity (Puerile Breathing).—Exaggeration of the vesicular murmur is noted over a lung which is the site of compensatory emphysema due to crippling of the opposite lung, over a circumscribed portion of a lung which is expanding vicariously to compensate for consolidation in an adjacent focus, in catarrhal inflammations of the smaller bronchioles, and during the dyspnea of imperfectly compensated cardiac disease.

Prolongation of the expiratory phase of the vesicular murmur accompanies hypertrophic emphysema and bronchial asthma. In these states the alteration in the phases of the sound is noted over both sides of the thorax; and in the case of asthma expiration is dotted with râles. Unilateral prolongation of the expiratory phase at an apex is suggestive of incipient pulmonary tuberculosis, particularly if noted at the left supraclavicular fossa.

Cog-wheel Breathing.—In certain diseases of the respiratory organs the respiratory murmur, and particularly the inspiratory phase of the murmur, occurs in a series of short gasps or jerks, closely simulating the sound emitted by a sobbing child. This jerking or cog-wheel modification of the vesicular murmur is a valuable sign of incipient phthisis. It is noted with far less constancy in hysteria, bronchial asthma, chorea, local catarrhal conditions of the bronchioles, in the pain of a fractured rib, pleurodynia, and pleurisy.

Bronchovesicular Breathing.—When encountered in an area of the thorax where it is not normally audible, this type of respiration points to a moderate degree of the same pathologic changes which produce frank bronchial breathing. It is a sign of p or incomplete consolidation, as in the early stage of pneumonia phthisis, or of a cavity or a solid tumor of the lung which covered by normal crepitant pulmonary tissue.

VOCAL RESONANCE

Vocal resonance is the transmission of inarticulate or artic late sound to the ear of the examiner upon auscultation of tl thorax during the act of phonation. In eliciting the phenomer of vocal resonance, the bell of the stethoscope is applied firml and evenly to the surface of the thorax while the patient directed to count "One, two, three," or to repeat the wor "ninety-nine" in a voice of uniform intensity and with the fa turned from the examiner. According to the aim of the examin tion, the patient repeats the words either aloud or in a whispe In either event, it is essential that the intensity of the voice r main uniform throughout the examination. In the course of tl examination the intensity of the sounds elicited from variou regions of the thorax should be studied, and the intensity up symmetrical regions should be carefully compared.

The intensity of vocal resonance presents regional variatio in the various regions of the thorax which correspond accuratel to the regional variations in the intensity of vocal fremitus in tl normal subject. As in the case of vocal fremitus, vocal resonan is elicited in its maximum intensity and purity upon auscultati of the larynx. Upon auscultation over the thyroid cartilage tl sound is so intense that it is frequently painful to the ea *laryngophony*. Moreover, the quality of the voice as appreciat upon auscultation is observed to have quite completely altered quality. It possesses upon auscultation a distinct nasal qualit which is rather closely simulated when the subject speaks wi the nares closed or with a membrane stretched tightly across tl lips.

The causes of the alteration in the quality of the voice a multiple, being due to the propagation of the vocal vibratio across the solid cartilages of the larynx instead of through fr air; to the fact that the propagation does not follow the directi of the molecular oscillations, but occurs perpendicularly to the and to a simultaneous mingling of the vibrations of the larynge cartilages with the vocal vibrations (Eichhorst).

n auscultation of the median line of the root of the nec nisternal notch, corresponding to the course of tl trachea, the vocal vibrations possess great clearness and intensity, *tracheophony,* but which does not equal the purity of the resonance as elicited over the larynx. Again, upon auscultation of the interscapular region at the level of the fourth dorsal vertebra, overlying the origin of the principal bronchi, the intensity of the resonance is also extremely pure, *bronchophony.*

Upon auscultation of the infraclavicular, mammary, axillary, infraaxillary, and infrascapular regions, regions of the thorax which overlie crepitant pulmonary tissue in which the larger bronchi are comparatively remote from the thoracic surface, the examiner appreciates in the normal subject only certain rumbling, inarticulate sounds, which arise in the vocal cords and are transmitted downward into the lung and to the thoracic surface by the air column of the trachea and bronchi and the pulmonary parenchyma.

As in the case of vocal fremitus, again, the intensity of vocal resonance depends upon the intensity and pitch of the voice, and upon the thickness and elasticity of the thorax; whence it follows that it is relatively less intense in the case of young children and in the female subject. In the aged subject, on the contrary, owing to wasting of the investing musculature of the thorax and to the progressive calcification of the bronchial cartilages, vocal resonance becomes progressively more intense with increasing age. In this class of subjects the sound very frequently assumes a quavering, nasal quality, analogous to egophony in younger subjects, on account of the natural quaver of the senile voice.

PATHOLOGIC VARIATIONS

As the intensity of vocal fremitus is modified by various lesions of the thoracic viscera, so also vocal resonance, the auscultatory equivalent of vocal fremitus, under similar circumstances exhibits variations which, in the main, are very similar and closely akin to the variations in vocal fremitus. In addition, in the presence of certain diseases of the bronchopulmonary system, vocal resonance exhibits definite and more or less pathognomonic modifications in its quality.

Diminution or Absence.—The intensity of vocal resonance is impaired in the presence of hypertrophic emphysema or compensatory emphysema, owing to the distention of the lung incident to these conditions. It is similarly diminished or abolished in the presence of pleural thickening, moderate pleural effusion, and bronchial stenosis. A pulmonary cavity containing fluid causes abolition of vocal resonance in the area in which it lies. While in the case of pleural effusion the usual finding is abolition of vocal resonance over the distribution of the fluid and exaggeration of the resonance above the limit of the fluid, yet in the presence of pleural adhesions which traverse the fluid effusion, it is possible for vocal resonance to be manifest in circumscribed portions of the thoracic surface overlying the effusion.

Increased Vocal Resonance.—Vocal resonance is increased by the same factors which cause an increase of vocal fremitus; namely, consolidations, pulmonary compression, and cavities with free bronchial communication. The various gradations of increased vocal resonance are designated by different names.

Bronchophony is a form of increased vocal resonance in which the transmitted voice is very distinctly audible, sounding as if it were very near the ear. However, the speech is not articulate as it is in the next ascending grade, pectoriloquy. Bronchophony points to dense consolidation, particularly to consolidation which overlies or is superimposed upon one of the principal bronchi.

Pectoriloquy, the transmission of the articulate voice upon auscultation, is evidence of a very dense consolidation overlying a principal bronchus, or of a pulmonary cavity or pneumothorax with free bronchial communication. With less frequency pectoriloquy is elicited upon auscultation of a portion of the lung which is compressed by a pleural effusion. In the cases in which the voice is transmitted with great distinctness it is very suggestive of a pulmonary cavity.

Whispering pectoriloquy, the transmission of the articulate whisper upon auscultation, represents the highest refinement of vocal resonance; and when elicited it is almost conclusive evidence of the presence of a pulmonary cavity with patent bronchial communication or of pneumothorax with bronchial fistula. In the normal subject the whispered voice is audible as such only upon auscultation immediately over the trachea. In extensive consolidations and conditions of pulmonary compression and collapse the whispered voice is frequently audible but is not articulate. An articulate whisper upon auscultation is very good evidence of a pulmonary cavity with free bronchial outlet.

Baccelli's Sign.—The whispered voice is transmitted through a serous pleural effusion, but is not transmitted through a purulent effusion. This sign is employed in differentiating between the two types of pleural effusion. While it is often a valuable means of amphorophony, or the cavernous voice. When elicited, it constitutes a reliable sign of a cavity containing an air column which vibrates simultaneously with the waves reaching it from the vocal cords.

ADVENTITIOUS SOUNDS

New, superadded, or adventitious sounds, which are engendered in pathologic states of the organs of respiration, may originate in the larynx, trachea, bronchi, lungs, or in the pleural cavity. They comprise various types of râles, the metallic tinkle, the succussion sound, the lung-fistula sound, and the pleural friction sound.

RÂLES

Râles are adventitious sounds which are generated in the larynx, trachea, bronchi, bronchioles, or pulmonary alveoli, as a result of interference with the free ingress and egress of air during inspiration and expiration. The lesion which is responsible for the production of râles, and which will be studied more in detail in connection with the detailed description of the various types of râles, may consist in a diminution of the lumen of the air passage by compression from without, or by turgescence of the mucous membrane within; it may be an obstacle imposed by the presence of serum, mucus, pus, or blood within the bronchial tubes; or it may consist in the cohesion of the walls of the terminal bronchioles and alveoli, which are glued together by a coating of tenacious secretion or fibrinous exudate.

Râles are classified primarily as *dry râles* and *moist râles*, depending upon whether or not moisture is, in the opinion of the examiner, the essential factor in their production.

Dry râles, or ***rhonchi***, which are invariably signs of stenosis, are produced by an obstacle to the free ingress and egress of air during respiration in the form of varying degrees of stenosis of the air passages, whether these passages be narrowed by the presence upon their walls of accumulations of tenacious secretion; whether the bronchial lumen be diminished by turgescent swelling of the mucous membrane; or whether the constriction be due to external pressure exerted upon the bronchial tube by bands of adhesions or enlarged thoracic viscera. The generation of the sound in each instance is caused by the passage of the inspired or expired air through a bronchial stenosis into a wider portion of the tube

beyond, with the consequent production of aerial whorls.
quality of the râle is dependent for this reason upon the size
bronchus in which it is generated and the diameter of the co
tion which is the causative agent.

Upon the acoustic influences of these two factors, it is p
to subdivide dry râles into *sonorous râles,* of low pitch, and
ing, often musical quality; and *sibilant râles,* of high pitc
hissing, whistling, or squeaking quality. Now, since only
moderate turgescence of the mucous membrane of the s
bronchi and bronchioles, or the presence of even minimal an

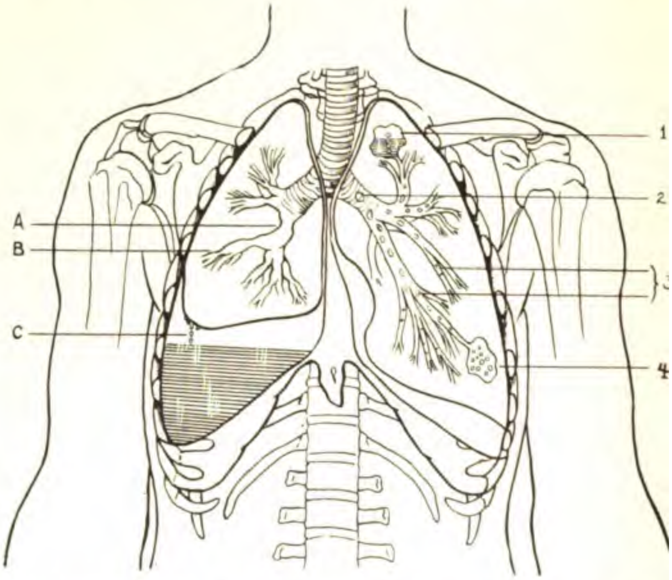


Fig. 67.—Illustrating the physical basis of pathologic physical signs upon
tion of the thorax. *A,* Sonorous râles; *B,* sibilant râles; *C,* metallic tinkle, or su
sound; *1,* gurgling or bubbling râles; *2,* coarse or mucous râles; *3,* fine or sub
râles; *4,* crepitant râles.

of secretion upon the walls of these tubes produces consid
stenosis in these small passages, and as their caliber is so l
that the distal portion of the tube cannot act in any consid
degree as a resonating chamber for the aerial whorls whi
called into being by the passage of the air through the cons
portion, sibilant râles are practically confined to the distri
of the smaller bronchi and bronchioles, while sonorous râl
produced only in the larger bronchi, the trachea, and the l

Dry râles are most frequently audible during inspiratio
frequently they are audible during inspiration and expiratio

very infrequently indeed are they audible during expiration alone. Dry râles, and this is true of moist râles as well, are frequently audible at some distance from the region of the thorax which overlies the site of their generation; but they always present a point of maximum intensity, which is situated over the portion of the bronchial system in which they are produced. When dry râles are encountered universally over the thorax, as is the case in bronchial asthma, it is commonly observed that sonorous râles are audible in the main during the first half of inspiration; whereas the sibilant râles come to the fore during the latter half of this act, as a result of the progressive penetration of the inspired air to the ultimate ramifications of the bronchial system. If the râles are audible also during expiration, the opposite sequence of events is observed. The preponderance of dry râles during inspiration and their frequent absence during expiration is doubtless in intimate relation with the rapidity of the aerial current through the bronchial system during the two phases of the respiratory cycle. In order that whorls of sufficient intensity may be produced in the air passages to produce sound, it is essential that the air pass the stenosis with a fair degree of rapidity, a physical state which obtains during inspiration to a much greater degree than it is present during expiration. This assumption is further substantiated by the fact that dry râles are frequently inaudible during tranquil respiration, to come out clearly during deep inspirations upon the part of the patient.

The relative pitch of dry râles is very readily appreciated even by the inexperienced examiner, the high pitched, squeaking, whistling sibilant râles contrasting markedly with the deep snoring of the sonorous râles. This variation in pitch possesses in this manner a distinct localizing value in diagnosis.

The intensity of dry râles varies with the force of the respiratory movements, with the degree of stenosis which engenders them, with the site of their production in relation to the thoracic wall, and with the thickness of this wall. When dry râles of considerable initial intensity are generated in the depth of the lung, where they are covered by thick masses of aerated pulmonary parenchyma, the intensity is greatly masked in the transmission of the râles to the thoracic surface. When, on the contrary, râles are generated in bronchial tubes of more superficial distribution, their intensity is as much greater upon auscultation, and they are frequently attended by rhonchal fremitus upon palpation.

Dry râles assume a special intensity and a distinct change in quality when they are engendered in a bronchus which terminates in portions of the lung which are the seat of compression and collapse, and when the bronchus terminates in a pulmonary cavity situated in the periphery of the lung. In this instance the pulmonary excavation acts as a resonating chamber for the bronchial vibrations, with the production of the *consonating râles* of Skoda. The râles in this case are attended by blowing, bronchial breathing and amphoric resonance upon percussion over the cavity.

Dry râles are audible in the earliest stages of bronchial inflammation as a result of moderate turgescence of the bronchial mucous membrane, prior to the pouring out of secretion into the lumina of the tubes, and in the paroxysm of bronchial asthma as a result of constriction of the bronchioles. The *bruit de drapeau* is a dry râle which is audible in certain cases of fibrinous bronchitis, and which is produced by the flapping back and forth of a fragment of adherent exudate or detached mucosa during inspiration and expiration. Sonorous râles are generated in the trachea in the presence of extreme compression of this tube by aneurysm, mediastinal tumor, or enlarged mediastinal glands, as well as in cicatricial stenosis of this tube. Similar râles occasionally arise in the larynx as a result of laryngeal diphtheria and in edema of the glottis.

Moist râles, produced by the passage of air through serum, mucus, pus, or blood in the larger bronchi, or by separation of the walls of the terminal bronchioles and alveoli, which have become adherent as a result of the presence of tenacious exudate, comprise the *crepitant râle*, the *subcrepitant râle*, the *mucous râle*, and the *gurgling râle*.

Crepitant Râle.—The crepitant râle is produced by the separation of the walls of the alveoli, which have become glued together by tenacious secretion during the expiratory recession of the lung. Hence, the râle is audible toward the termination of inspiration.

The crepitant râle is the finest of all râles. Its quality has been compared by Laennec to the series of sharp cracklings which ensue upon throwing a pinch of salt upon a heated stove. Williams compares the quality of the râle to the sound which is produced upon rolling a lock of hair between the thumb and finger when held near the ear. Both comparisons are inexact, as moisture is an essential element in the production of the crepitant râle. The quality of the râle is aptly imitated upon moistening the tips of the thumb and forefinger, pressing them together, and separating them while the hand is held near the ear. The quality of the crepitant râle is readily confused with the pleural friction sound, when the latter possesses a minor intensity. The diff ential points between the two sounds are considered in connect with the pleural friction sound in a subsequent paragraph.

The crepitant râle is not infrequently audible upon auscul tion of the pulmonary bases in the normal subject who has pas a night breathing tranquilly in the dorsal decubitus. In t instance, however, a few deep inspirations serve to fully infl the alveoli and to abolish the râle in this area. As this râl dependent upon a wet lung for its production, it is natural t in the diseases in which it is present it is encountered over bases in the majority of cases. In all cases in which crepit râles are encountered over the apices of the lungs, the progno significance is grave; and if the râles are localized in this area the exclusion of other portions of the lungs, it is very suggest of phthisis as the underlying lesion.

The crepitant râle is detected during the first stage of fibrin pneumonia, constituting the *crepitus indux* of this disease. It also audible over the bases of the lungs in the presence of infl mation of the mucous membrane of the terminal bronchioles pulmonary alveoli in catarrhal pneumonia or capillary bronchi and it is audible in the same regions in the presence of pulmon edema, hemorrhagic infarction of the lung and in partial atele sis. During the evolution of chronic ulcerative phthisis, and w less constancy in the stage of incomplete consolidation in fibrin pneumonia, there is occasionally audible a fine, high-pitched cre tant râle, the *mucous click*. As in the case of all crepitant râ the mucous click is elicited toward the completion of inspirati when the inspired air has attained the ultimate ramifications of bronchial system.

Subcrepitant Râle.—The subcrepitant râle is a moist râle trifle coarser in quality than is the crepitant râle. It is produ by separation of the walls of the terminal bronchioles which h become adherent as a result of a coating of tenacious secret or exudate; hence, it is audible during both inspiration and piration. The subcrepitant râle occurs during the period resolution of fibrinous pneumonia, in which disease it con tutes the *crepitus redux;* during the evolution of acute and chro catarrhal bronchitis; and in the presence of bronchial inflammat in catarrhal pneumonia, pulmonary infarction, and pulmon edema.

Mucous Râle.—Mucous râles are generated in the lar chi and in pulmonary cavities. They are audible during tranquil respiration, and are devoid of pathologic significance. limited number of moist râles, encountered over the apical port of the thorax, on the contrary, is of grave prognostic significar if they are persistent and are encountered in the same area u consecutive examinations. Circumscribed moist râles in this reg are very suggestive of phthisis; but Rosenbach has directed att tion to the extreme importance of not mistaking muscular sou in this region for moist râles, particularly in subjects with pow fully developed pectoral muscles.

Again, as in the case of acute and chronic catarrhal bronch and bronchial asthma, moist râles are distributed universally o both sides of the thorax, and are frequently in the latter dis of such intensity as to be audible without the aid of the stethosc

The number of the râles is largely dependent upon the amo of the pathologic secretion which is present in the bronchial syst upon the energy of the respiratory movements, and upon the of the causative lesion with reference to the thoracic surface. W the bronchial secretions are unduly abundant and when the resp tory action is very pronounced, if the lesion is deeply seated in central portion of the lung, there will be a minimal number râles demonstrable on account of the interference with their duction which is offered by the intervening areas of aerated monary tissue.

It occasionally happens that in a given case moist râles so abundant that the character of the respiratory sounds are termined with difficulty, as the quality of the respiratory mur is effectually masked by the predominance of the râles. In s event Lasègue recommends auscultation of the respiratory mur immediately following vigorous coughing efforts upon the par the patient, at which time the râles are temporarily in abeya and the quality of the respiratory murmur may be studied.

The *size* of râles is dependent upon the character of the bron secretions, the energy of the respiratory movements, and the of the lesions with reference to the thoracic wall. In respec their size, râles are divided into large râles, medium râles, small râles. Large and medium râles are only encountered in presence of fluid secretions in the lumina of the larger bron while small râles are engendered by the presence of fluid in bronchi of smaller caliber. In the case of the large and medi ed mucous râles, the intensity of the râles is in direct propor he respiratory movements and the proximity stion to the thoracic wall.

The *uniformity* of the râles which are encountered should be studied in every case in which moist râles are encountered. In this manner the student determines whether he is dealing with only one type of râle or with various types of râles, and from this information he deduces conclusions as to the portion of the bronchopulmonary system which is at fault. Even in the case of râles of the same character it is occasionally possible to detect differences in the quality and pitch of the râles. Thus, Laennec held that the crepitant râle of lobar pneumonia is uniformly more intense and of higher pitch than is the crepitant râle of pulmonary edema.

The *time of appearance* of râles with reference to the events of the respiratory cycle is of aid in determining the character of râle with which the examiner is confronted, and serves as a basis for conclusions as to the site of the lesion of the bronchial system.

Thus, the crepitant râle and the great majority of gurgling râles become apparent toward the completion of inspiration; whereas the subcrepitant râle is demonstrable during both inspiration and expiration. Similarly the mucous râle generated in the larger bronchial tubes is audible during both phases of the respiratory cycle. The study of the time of appearance of râles should be abetted by the careful study of the quality of the râles in question.

The *intensity* of râles depends upon the site of their production, the number of the râles, the size of the râles, and the state of the superimposed pulmonary parenchyma. Râles which are developed in the superficial bronchi are naturally more intense than are râles developing in bronchi of the central portions of the lungs. However, the intensity of râles developing in the depths of the lungs is influenced by the state of the pulmonary parenchyma which is superjacent to these bronchial tubes, and as this is consolidated, compressed, or excavated by phthisis, they assume an undue intensity and special qualities which will be described in a subsequent paragraph. When râles are very abundant and universally distributed throughout the lungs, their intensity is augmented by their number alone, not infrequently to the extent that they are audible without the aid of the stethoscope or, indeed, at some distance from the patient. The size of the râle exerts a perceptible influence upon its intensity. The large and medium râles of the larger bronchi possess a greater intensity than do the small râles which are generated in the smaller bronchial tubes.

The *quality* of the moist râle is more difficult to seize and to define than in the case of dry râles. However, it is upon this difference in quality between dry and moist râles that the examiner bases his conclusions as to whether he is dealing with a dry with a wet lung. The bubbling quality of gurgling râles is readil appreciated and the incidence of the crepitant and subcrepita râle at different periods of the respiratory cycle serves as a check i some measure upon these râles.

Moreover, when moist râles are generated in pulmonary tiss which is in close proximity to pulmonary excavations, or not i frequently even when the stomach or colon is excessively distende the râles take on a musical quality which is closely akin to t quality of the consonating râles of Skoda.

Consonating moist râles possess the musical quality to a mark degree; and, as either pulmonary excavation with bronchial comm nication or pulmonary relaxation and collapse, furnish excelle conducting media, these râles are transmitted to the surface of t thorax with undue intensity. These râles are attended by bronch breathing, exaggeration of vocal fremitus and vocal resonan and by the amphoric percussion sound.

THE METALLIC TINKLE (GUTTA CADENS; FALLING-DROP SOUND)

During auscultation of the thorax which is the seat of hydr hemo- or pyo-pneumothorax, and with less constancy duri auscultation over a large pulmonary cavity which contains flui a sound is occasionally audible during inspiration and expirati which resembles that which is produced by drops of water falli from a height upon the surface of water contained in a ciste The sound has a hollow, echoing, metallic quality; it is mo frequently present during inspiration, though occasionally hea during expiration as well; and it is frequently brought out changes of posture, speaking, coughing, or by jarring or shaki the subject.

The physical genesis of the falling-drop sound is obscure, a several theories have been evolved in the attempt to explain t mode of production of the sound in the various pathologic cas in which it has been encountered. Laennec originally attribut the sound to the dripping of fluid from the retracted inferi border of the lung to the surface of an accumulation of flu occupying the lower portion of the pleural cavity. Baas oppos this theory, asserting that droplets of fluid which are formed up perior walls of a cavity have a tendency to glide along t of the cavity, instead of falling abruptly from these sup rior regions. Leichtenstern, however, encountered a very pure falling-drop sound in a case of pyo-pneumothorax, which was only demonstrable when the patient passed from the dorsal decubitus to the sitting posture. Upon autopsy the case presented villosities of the pleural surface, which were immersed in the fluid with the patient in the dorsal decubitus, and which dripped fluid when the patient was placed in the upright posture.

Another theory of the mode of production of the metallic tinkle is the assumption of the bursting of bubbles which are formed upon the surface of an accumulation of fluid in the pleural cavity. Force is lent to this explanation in certain cases by the fact that the sound is elicited immediately following the succussion sound upon jarring the subject.

Debove and Trémolières find a striking similarity between the quality of the falling-drop sound and that of the Hippocratic succussion sound when the latter is elicited by gentle agitation of the patient. These authors believe that the metallic tinkle is due neither to the dripping of fluid from the retracted borders of the lung nor to the bursting of bubbles upon the surface of the pleural fluid; but that it is due to the generation of a light wave upon the surface of the fluid enclosed in the pleural cavity, the latter acting as a resonating chamber for the slight sound which is so generated. They hold that the rhythmical diaphragmatic movements are alone sufficient to call into being the causative waves in the intrapleural fluid.

It has also been asserted that the metallic tinkle is only generated in connection with accumulations of fluid in the pleural cavity when there is a patent bronchial communication with the pneumothorax; and that the sound is to be attributed to the bursting of a bubble which has formed at the orifice of the bronchopulmonary fistula at the moment of full inspiration.

HIPPOCRATIC SUCCUSSION; (SPLASHING SOUND)

In the presence of hydro- hemo- or pyo-pneumothorax, when the upper portion of the trunk is abruptly jarred or shaken, the ear of the examiner meanwhile being applied closely to the thoracic wall, a distinct sound of splashing is frequently audible, which is analogous to that which is produced by suddenly moving a partially filled cask. The succussion sound is a reliable sign of the presence of air and fluid in the pleural cavity; hence it is absent in pleurisy with effusion. The succussion sound arising within the pleural cavity should not be confused with simil splashing sounds which are generated within a dilated stoma

While in the case of large pulmonary cavities containing and fluid the physical conditions are ideal for the generation Hippocratic succussion, it is rare that these excavations are favorably situated with reference to the thoracic surface th succussion sounds are appreciable over them.

THE PLEURAL FRICTION SOUND

In the normal subject, as a result of the smooth, polished su face of the visceral and the parietal pleura, moistened by a moder amount of serous fluid, these membranes glide noiselessly over ea

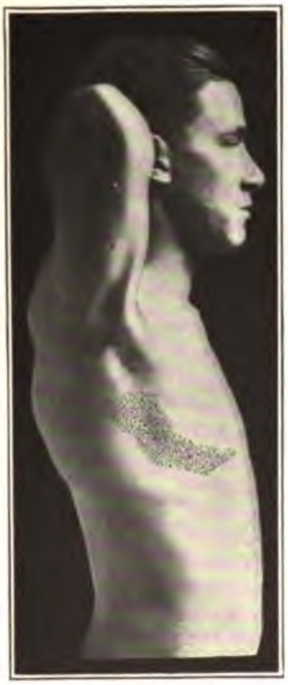


Fig. 68.—Usual site of pleural friction sound.

other during the respiratory movements. During inflammation the membrane, however, and as a result of the excessive extracti of the body fluids which accompanies prolonged diarrhea a profuse hemorrhage, as well as in the presence of miliary tub ulosis and carcinomatous infiltration of the pleura, there erated a pleural friction sound, which is audible upon ausc tation of the surface of the thorax. Jürgensen and Waldenburg would make a distinction between pleuritic friction, which is due to inflammatory processes of the pleural membrane, and pleural friction, which occurs with roughening of the membrane by the nodules of acute miliary tuberculosis, carcinomatous infiltration of the pleura, and osseous excrescences of the ribs, which they do not regard as essentially inflammatory in origin. As the pleural membrane is extremely prone to undergo inflammatory changes in the presence of even minor grades of irritation, it is not improbable that all of these conditions are attended by pleuritis of varying intensity.

The pleural friction sound presents extensive variations in its quality and intensity. In cases of slight pleural involvement it possesses a very light, grazing sound, analagous to that which is produced when the finger lightly strokes a silken fabric. In the presence of more severe pleural inflammation, the sound is very similar to that which is produced upon walking upon dry, new-fallen snow, or to the creaking of a new leathern saddle, which has given rise to the name of the *bruit de cuir neuf.*

While in many instances the pleural friction sound is a continuous sound, appreciated throughout the greater duration of the respiratory phase in which it is audible, in other cases it presents one or more interruptions, as if the roughened pleura were successively surmounting obstacles during the excursion of the lung.

The intensity of the pleural friction sound is in direct proportion to the intensity of the respiratory movements, to the extent of the pleura which is diseased, and to the severity of the inflammatory process. In cases of very limited pleural involvement the sound is very faint, requiring a trained ear for its detection. In other cases the sound is so intense as to be audible without the aid of the stethoscope, and is accompanied by distinct pleural friction fremitus upon palpation. Voluntary acceleration of the respiratory rate and increased depth of inspiration serve to increase the intensity of the sound. The intensity of the friction sound may be accordingly increased by the exercise of pressure in the intercostal spaces in the area in which it is encountered. Occasionally the friction sound is temporarily suppressed after several deep inspirations, probably as the result of smoothing out of the rugosities of the membrane, as the sound reappears upon resuming respiration after a momentary suspension.

The localization of pleural friction naturally corresponds wit the distribution of the inflammatory disease of the pleura. It i encountered most frequently in the lower axillary and infra axillary regions, where it is most frequently observed along a ascending and descending plane. When, in these areas, the frictio seems to occur in a transverse direction, it is suggestive of the limitation of the vertical excursion of the lung by pleural adhesions Pleural friction is not frequently encountered over the apica portions of the lungs, as these regions enjoy only a limited rang of mobility during the respiratory movements; but when pleura friction is encountered here, it is very suggestive of tuberculou lesions which are complicated by a fibrinous pleurisy.

The duration and persistence of pleural friction are subject t wide variations. During the course of serofibrinous pleurisy th friction sound appears prior to the development of the effusion; i disappears with the advent of the effusion; and not infrequently i again becomes audible with resorption of the effusion, occasionall to persist thereafter during the entire life of the individual. I cases of abortive acute fibrinous pleurisy, the sound may be presen upon one examination, to disappear immediately thereafter. Whe pleural friction develops in the supra- and infraclavicular region in connection with chronic ulcerative phthisis, the sounds commonly persist throughout the course of the disease.

As pleural inflammation very frequently complicates diseases o the lungs which are attended by râles, it is natural that pleura friction and râles of various types are frequently present in th same subject; and in many instances their differential diagnosis i attended by some difficulty. In this connection it is to be recalle that upon exerting pressure with the stethoscope in the intercosta spaces the intensity of pleural friction is commonly accentuated while this maneuver is without influence on the intensity of râles Moreover, râles are more continuous than is the case with pleura friction; râles change in character or transiently disappear afte coughing attacks; and compression of the thorax in the presenc of pleural disease is acutely painful, while similar compression o the chest in pulmonary disease unattended by pleurisy is provocative of only slight pain if indeed there is any pain.

THE LUNG-FISTULA SOUND

he presence of hydropneumothorax with a bronchial com
n opening into the pleural cavity below the level of th
[1] first described the lung-fistula sound. The sound i

manifested by a series of bubbling, gurgling sounds upon auscultation of the diseased side under suitable conditions. When, under proper maneuvers, the aerial content of the pleural cavity is rarefied in the region superjacent to the fluid, during inspiration a portion of the inspired air enters the cavity below the level of the fluid, and in ascending to the surface, engenders bubbles which produce the gurgling, lung-fistula sound. Unverricht noted the sound following partial aspiration of a hydropneumothorax, the superjacent air in this instance being rarefied by the evacuation of the fluid.

In Riegel's case the sound appeared without aspiration of the fluid. In this case, as soon as the patient was placed in the sitting posture, he expectorated a considerable quantity of the pleural fluid, which passed by way of the bronchial fistula. Following this partial evacuation of the fluid, the balance between the aerial pressure in the bronchial system and in the pleural cavity was disturbed; and during inspiration a portion of the inspired air entered the pleural cavity below the level of the fluid, and in its ascent through this latter engendered the lung-fistula sound.

The sound may be elicited without aspiration or expectoration of the fluid by the procedure of Meezenbrock. This author has the patient assume the lateral decubitus, lying upon the side of the disease. He then seizes the dependent side of the thorax between the hands, and by compression evacuates a portion of the pleural fluid into the air passages through the fistulous opening. When the patient assumes the sitting posture and the pressure is gradually released from the diseased side, the air penetrates into the pleural cavity through the fistulous opening and produces the lung-fistula sound

CHAPTER VI

THORACOMETRY, CYRTOMETRY, AND THORACENTESIS

Thoracometry, or mensuration of the thorax, is employed to determine at consecutive examinations variations in the total circumference of the chest; to determine the presence of unilateral bulging or retraction of the thorax; and to estimate the total expansion of the chest.

In the determination of the total expansion of the thorax the difference between the circumference of the chest during complete expiration and during complete inspiration is taken, the difference between the two measurements taken at the level of the nipples indicating the total expansion or vital capacity of the thorax. A total expansion of three to four inches obtains in the average adult male subject, though slight discrepancies below or above these figures are not to be considered pathologic.

In the determination of unilateral variations in the size of the two sides of the thorax it is customary to measure from the midspinal line to the midsternal line upon each side and note any discrepancy in the two measurements. Allowance must be made for the fact that the right half of the thorax is normally slightly larger than is the left half. In making all measurements of the thorax the common tape measure is the appliance of choice, save in calculating the various diameters of the thorax, when a specially devised calipers is employed.

In determining the anteroposterior and transverse diameters of the thorax the calipers is used. In the estimation of the anteroposterior diameter one point of the instrument is placed over the midspinal line and the other over the midsternal line, and the measurement is read off on the scale of the instrument. The transverse diameter of the thorax is determined by applying a point of the calipers to each midaxillary line and reading the diameter as indicated upon the scale.

Cyrtometry, the determination of the curves of the surface of the thorax, is practiced by applying the cyrtometer accurately to the surface of the thorax. The cyrtometer consists of two pieces of flexible metal connected at one end by a hinge. In practicing metry of the thorax the hinge is placed over the midspinal line, and the blades of the instrument are accurately moulded to the surface of the thorax. Upon removal of the instrument a tracing may be made, showing the shape of a cross-section of the thorax, and revealing any unilateral variations of the two sides of the chest.

Thoracentesis, the aspiration of fluid from the pleural cavity, is usually preceded in practice by *exploratory puncture* of the pleural sac. The latter maneuver is frequently performed in order to establish the diagnosis of fluid in the pleural cavity and for the purpose of securing a specimen of fluid for microscopic and bacteriologic examination.

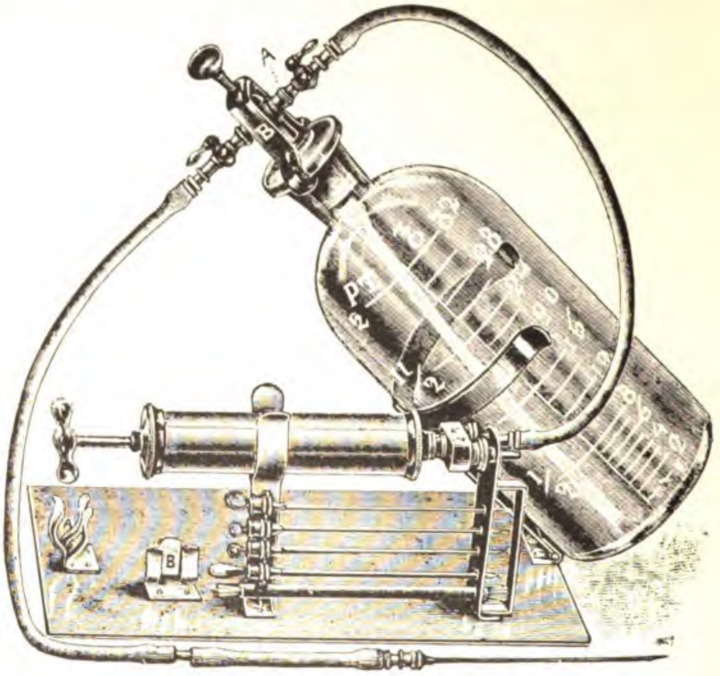


Fig. 69.—Potain's aspirator.

In the performance of the slight operation of exploratory puncture of the pleura, a large hypodermic syringe equipped with a heavy needle should be employed. Aseptic precautions should be strictly observed throughout the procedure in order to avoid the introduction of infective organisms into the pleural cavity, thus converting a serous into a purulent effusion. The pleural cavity may be entered in the scapular line in the seventh intercostal space or in the midaxillary line in the sixth interspace with equally satisfactory results. In securing a specimen by explor tory puncture, only a few cubic centimeters should be withdraw which should immediately be transferred to a sterile test tu for microscopic and bacteriologic study.

As exploratory puncture is performed for purposes of dia nosis, so aspiration is practiced for therapeutic purposes. In t practice of aspiration a large needle, connected by rubber tubi with a closed jar from which the air has been exhausted, is i serted into the pleural cavity beneath the level of the fluid a the fluid is gradually drawn off into the vacuum. In the ins tion of the needle, the intercostal artery, which courses along t lower border of the rib, should be avoided.

During aspiration it is not advisable to withdraw entirely t fluid which occupies the pleural cavity at a single sitting. Mo over, if at any time during the aspiration the patient complai of distress, the procedure should be immediately suspended.

CHAPTER VII

RADIOGRAPHIC DIAGNOSIS

By Dudley E. Mackey, B.S., M.D., New York.

The purpose of this chapter upon radiographic diagnosis is to treat only of the x-ray with reference to its use as a link in the chain of clinical data, as a means of examination which will aid the student of medicine or the physician in rendering a diagnosis. The principal aim throughout will be to present as nearly as possible in the space employed definite facts which are used and brought out in radiographic examination of various regions of the human body. Obviously it is impossible in this place to deal with technic and the use of apparatus.

Remarkable progress has been made in the advance of radiographic diagnosis since its introduction in 1895, and this has been especially made so by the invention of the Coolidge tube, which has given such good service in the recent war.

In this chapter the subject matter will embrace radiographic diagnosis of bones and joints; the head with the accessory sinuses, mastoids, and teeth; the thorax, gastrointestinal tract, and urinary tract.

BONES AND JOINTS

In dealing with the bones and joints, radiography is of value in enabling us to determine whether a fracture is present, the position and number of the broken parts, the best method of correction, and serves as a means of observation after a retentive dressing has been applied.

Radiographically, fractures are classed as simple and as comminuted. The simple fracture may be further subdivided according to the direction of the fracture. Fractures may be studied with the x-ray either fluoroscopically or by the plate method. For a permanent record the plate method is preferable. If the plate method is employed, two views of the part examined should always be required, while in the examination of joints a diagnosis should not be attempted without the use of stereoscopic plates. The latter method of examination will sometimes bring

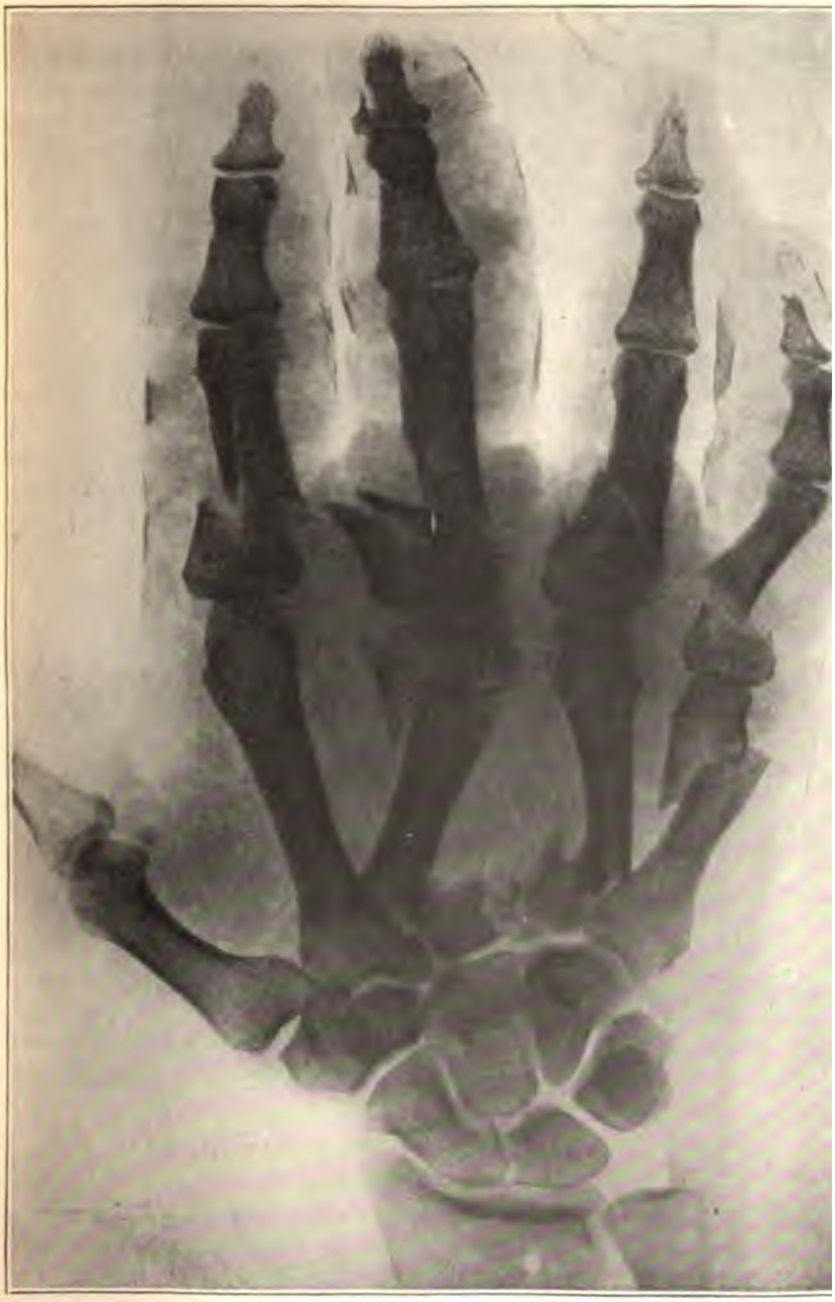


Fig. 70.—Compound-comminuted fractures of phalanges and metacarpal of hand.

out fine, hair-line fractures which would be overlooked on a flat plate.

In the examination of a fracture, before rendering a diagnosis, it is necessary to observe the area involved, the shaft, tissue surrounding the fracture, the bone or bones involved, its condition, displacement, position, type of fracture, and number of fragments. If the case is one of long standing with bone changes, search should be made for sequestra, giving their size and position. Observation should also be made as to whether the joint surfaces are invaded by the fracture. Under no circumstances should the splints or bandages be removed during radiography without the consent of the attending surgeon.

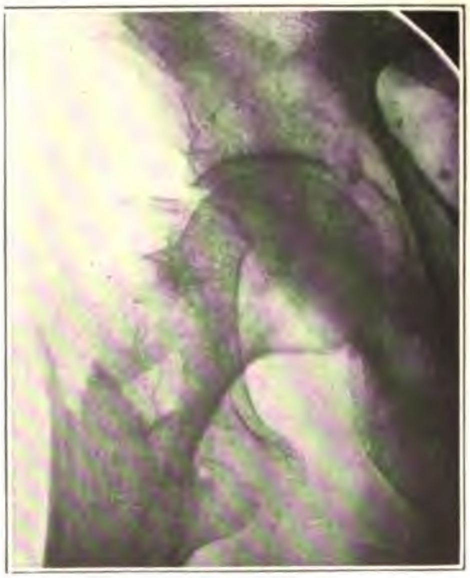


Fig. 71.—Stellate fracture of great trochanter of femur.

The student who would become proficient in the interpretation of plates must gain a thorough knowledge of the bony skeleton, together with the study of normal radiography. It is essential that he should study the normal adult subject. The study of the ossification of the epiphyseal lines radiographically is very important, and any standard work upon anatomy will give this information. Comparative plates of both right and left will sometimes differentiate between a fracture and an epiphyseal separation in a doubtful case.

Fractures of the skull are in some cases very difficult to di
nose, because of the presence of sutures and blood vessel ma
ings. The diagnostician should be very careful not to mista
a suture line for a fracture. Indeed, a fracture of the skull m
not give marked evidence of the break in continuity, yet m
cause injury to the blood vessels, nerves, and brain substan

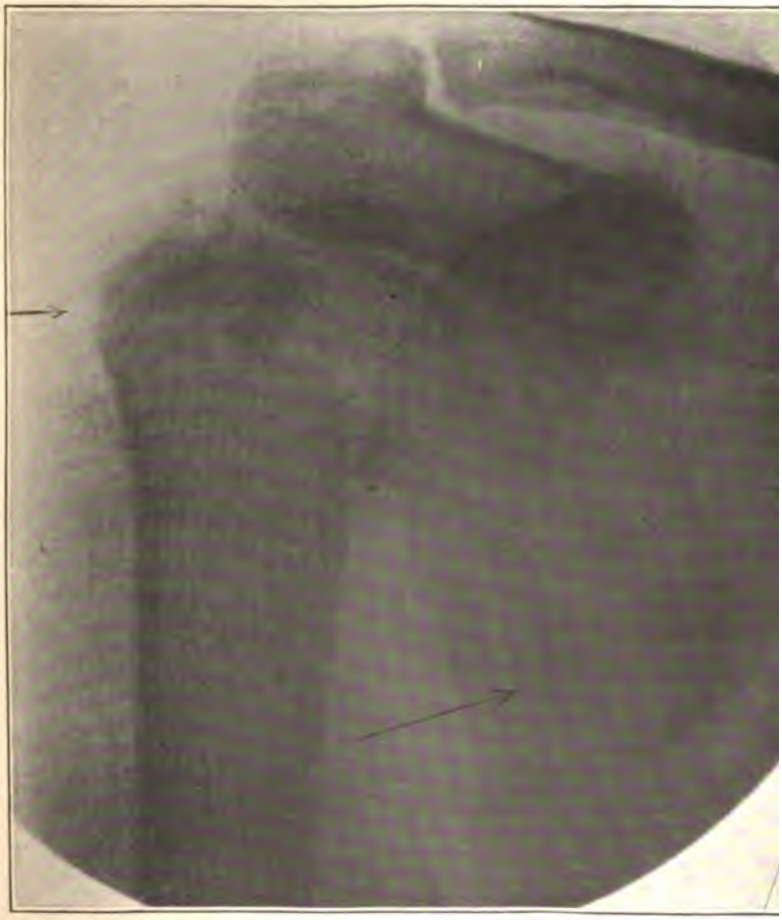


Fig. 72.—Impacted fracture of head of humerus with separation and displacement of h

The diagnosis of fracture of the skull should not be attempt
without the use of stereoscopic plates. With these for obser
tion, a thorough examination of the skull should be made, keepi
ever in mind the fact that blood vessel markings and sutures ha
been mistaken for fractures.

In dealing with suspected fractures of the skull the examiner should systematically examine the contours of the inner plate for any break in continuity. Here especially the student should be familiar with all the shadows and markings which appear upon the normal plate. The technic is very important in the correct reading of plates in head injuries.

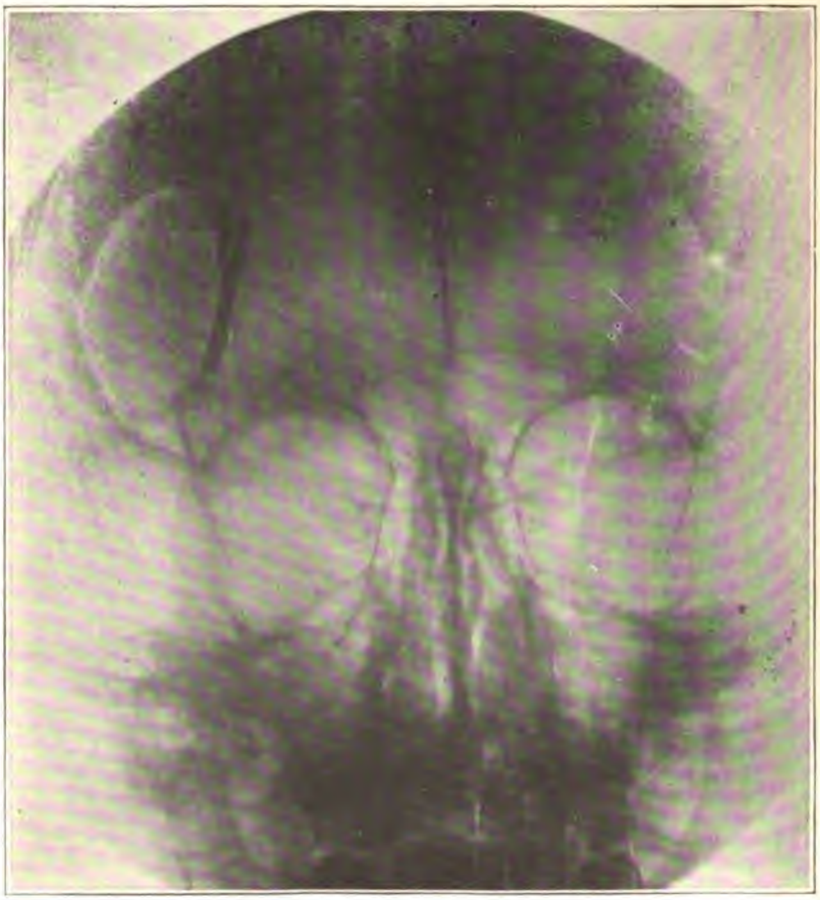


Fig. 73.—Depressed fracture of the skull.

Radiographic diagrams of the spine should always be made, if possible, from stereoscopic plates, together with a lateral view. Fluoroscopic examination of the spine is of very limited value, except in certain cases of the cervical spine or lumbar region. A careful examination of each vertebra should be made before rendering a decision, keeping the normal picture ever in mind.

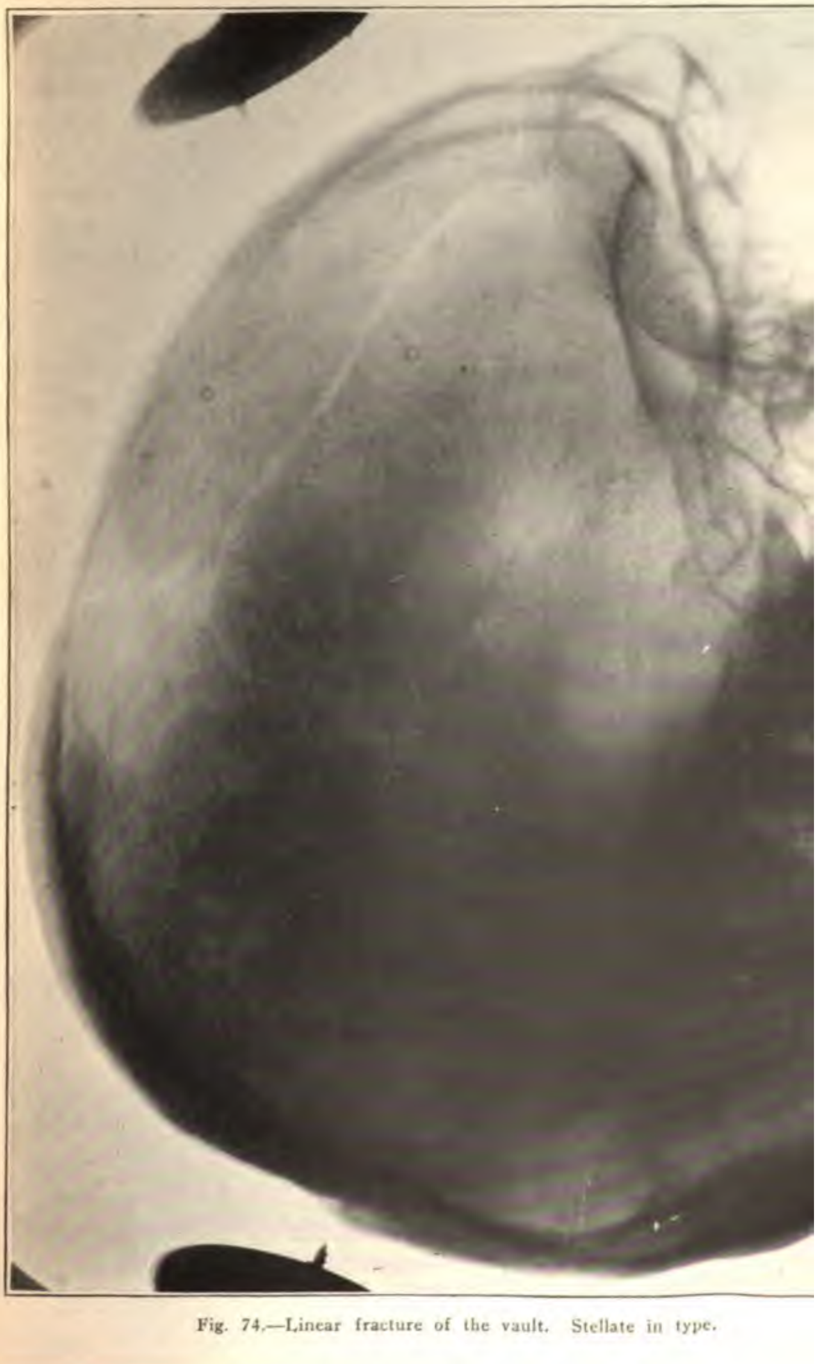


Fig. 74.—Linear fracture of the vault. Stellate in type.

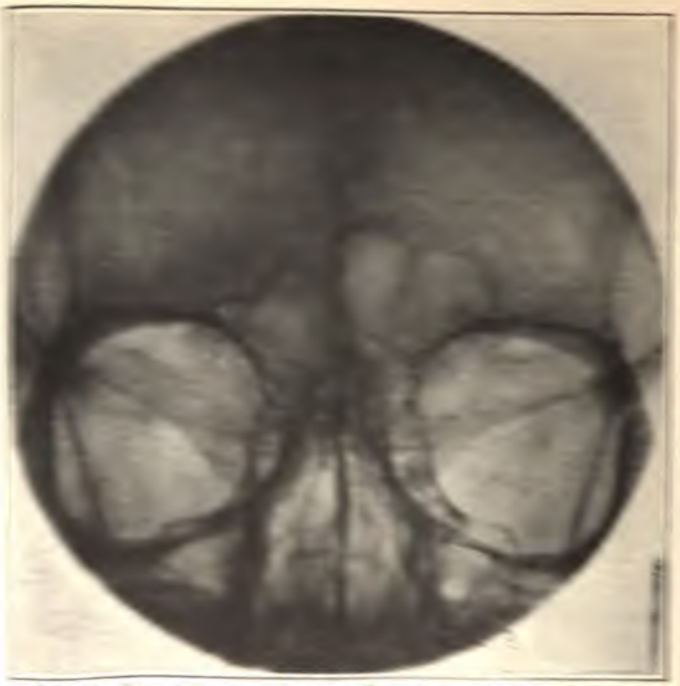


Fig. [illegible].—Linear fracture of the skull involving the frontal sinus.

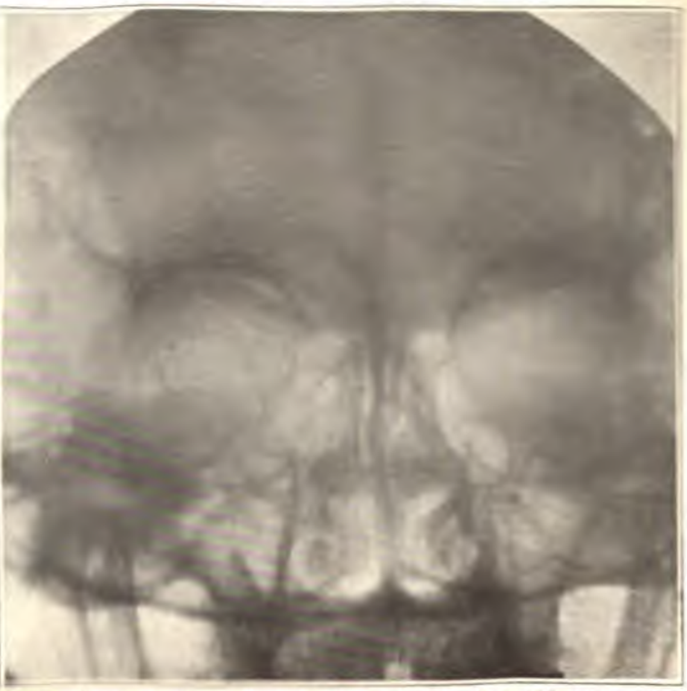


Fig. 76.—Accessory sinus. Frontals clear, ethmoids clear, and antrum clear.

Pathologic conditions found in the spine comprise lesions involving the bodies of the vertebræ, and lesions involving the articulating surfaces. Among the lesions of the articulating surfaces we class arthritis, infectious and hypertrophic, and th beginning of tuberculosis. Lesions may have their inception i the articulating surface and invade the body, notably tuberculosis in the second stage, neuropathic conditions, and fractures We may have new growths and osteomyelitis, both of which ma involve the body of the articulating surface.

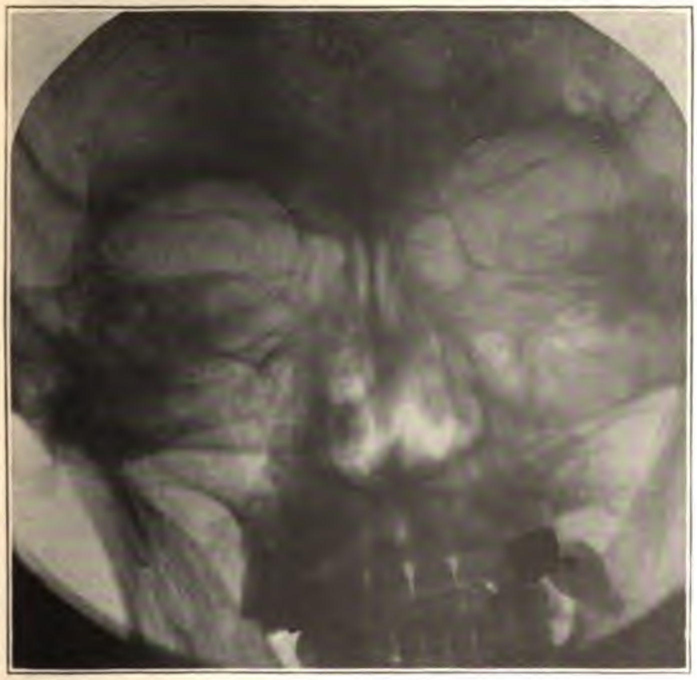


Fig. 77.—Absence of frontal sinus.

Tuberculosis is a bone-destroying process, not a bone producer In tuberculosis of the spine the anterior portion of the body becomes softened and gives way, becoming characteristic in appearance, triangular in shape, with the apex anteriorly and th base of the triangle posteriorly, leading in time to tubercula kyphosis.

Differential diagnosis between tuberculosis of the spine, fracture, neuropathic conditions, and bone lesions:

are there evidences of infiltration? By the use of the followi classification, the student will be enabled to study and analy the radiographic findings:

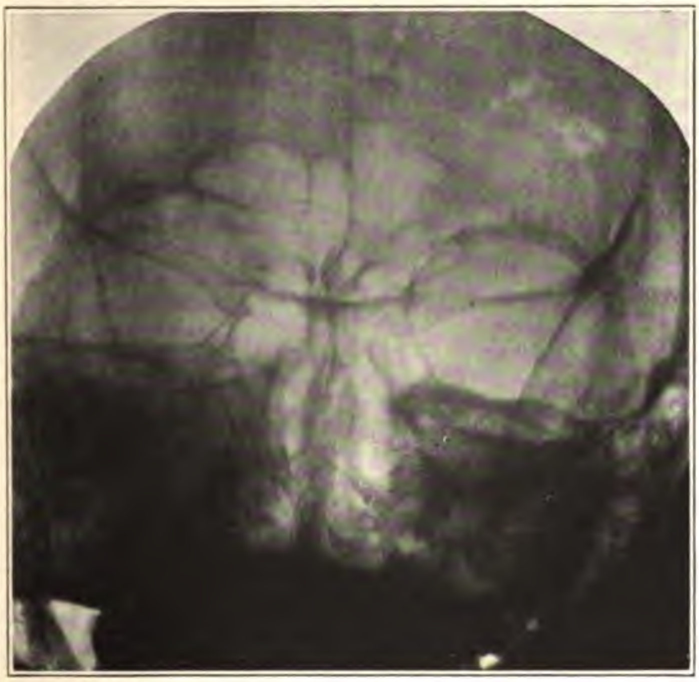


Fig. 79.—Frontal sinus clear, ethmoids clear, both antra cloudy.

LESION	ORIGIN	BONE PRODUCTION	INFILTRATION	TYPE
Exostosis	Shaft	Present	Absent	Benign
Osteoma	Cortex	Present	Absent	Benign
Osteosarcoma	Cortex	Present, with destruction	Present	Malignant
Periosteal sarcoma	Periosteum	Present in soft tissues	Present	Malignant
Giant cell sarcoma	Medulla,	Absent	Expected	Benign
Cyst	Medulla Cortex	Absent	Expected	Benign
Ossifying hematoma	Periosteum, hemorrhage	Present	Absent	Benign
Periostitis	Periosteum, inflammatory, luetic	Present	Absent	Benign
Osteomyelitis	Shaft	Present, with bone destruction	Present	Benign
rcinoma	Medulla, near nutrient artery	Absent	Present	Malignant

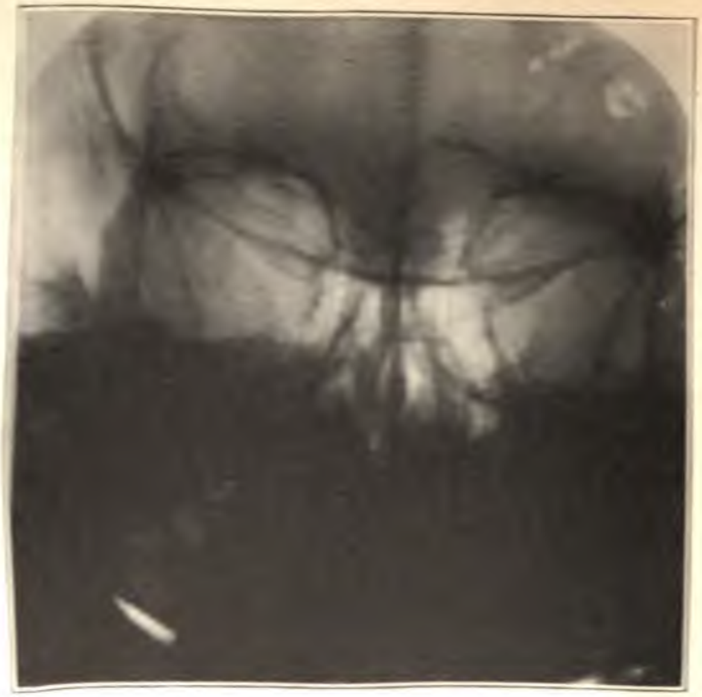


Fig. 50.—Absence of one frontal sinus. Ethmoids clear, both antra cloudy.

ARTHRITIS

In arthritis we have to deal with three structures; namely, synovial fluid, synovial membrane, and cartilage. The diagnosis, therefore, must be made upon the changes encountered in the above structures. Radiographically, we may classify arthritis as follows:

ARTHRITIS	AGE	FLUID	BONE	SYNOVIAL MEMBRANE	DESTRUCTION OF CARTILAGE
Polyarthritis	Any	Increased	Negative	Swollen	Absent
Infectious, first stage	Adult	Increased	Negative	Swollen	Absent
Infectious, second stage	Adult	Absorbed	Atrophy	Swelling decreased	Absent
Infectious, third stage	Adult	None	Atrophy	None	Eroded
Atrophic	Early, middle	Absent	Atrophy	None	Absorption
Hypertrophic	Middle, old age	Absent	None	None	Present with exostosis
Joint tuberculosis	———	Absent	Hazy	Thickened	None
Tuberculosis subsides	———	Absent	clear but eroded	None	Present
Syphilitic	———	Present	Periostitis	Thickened	None
Charcot	———	Present	Destruction	Thickened	Destruction, new bone
Gonorrheal	———	Present	Change and exostosis	None	Ankylosis patellar cartilage

THE HEAD

The radiographic study of the head should be made from stereoscopic plates, as the complexity of the shadows is such that clear reading of the plates cannot be made from flat plates.

One of the most difficult and one of the most frequently overlooked conditions is the diagnosis of fracture of the skull. A stated, blood vessel markings and sutures have frequently bee mistaken for fracture lines.

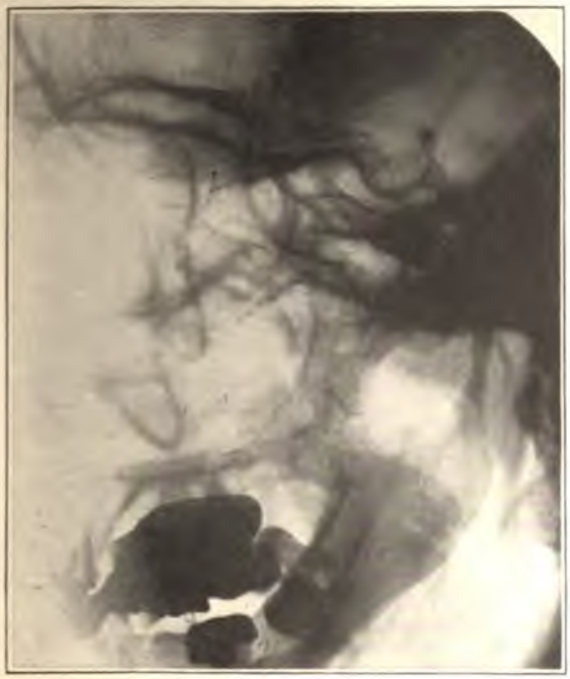


Fig. 81.—Sella turcica well defined—normal sphenoidal sinus clear.

Fractures of the skull are generally of two classes; namel linear, and depressed. The depressed fracture, which is cause by a blow from without, may affect the inner table only, and i is most important that this type of fracture should be detecte Without stereoscopic plates it is not apt to be clearly defined.

The study of the *mastoid area* is of the utmost importance, and requires for proficiency an extensive study in the interpretation o shadows. The size of the cells, which do not appear before th age of five years, has an important bearing upon the prognosi Both right and left mastoids should be made for comparison, a they are generally similar in structure. It is very difficult for the student to acquire a knowledge of mastoid conditions from printed matter, as the observation of a large number of plates and a study of the operative findings is necessary for the attainment of proficiency in this field of work. The same is equally true of the accessory sinuses, since when pathology is present, the accuracy of the diagnosis depends entirely upon the technic of making the plate and the comparison with the normal shadow.

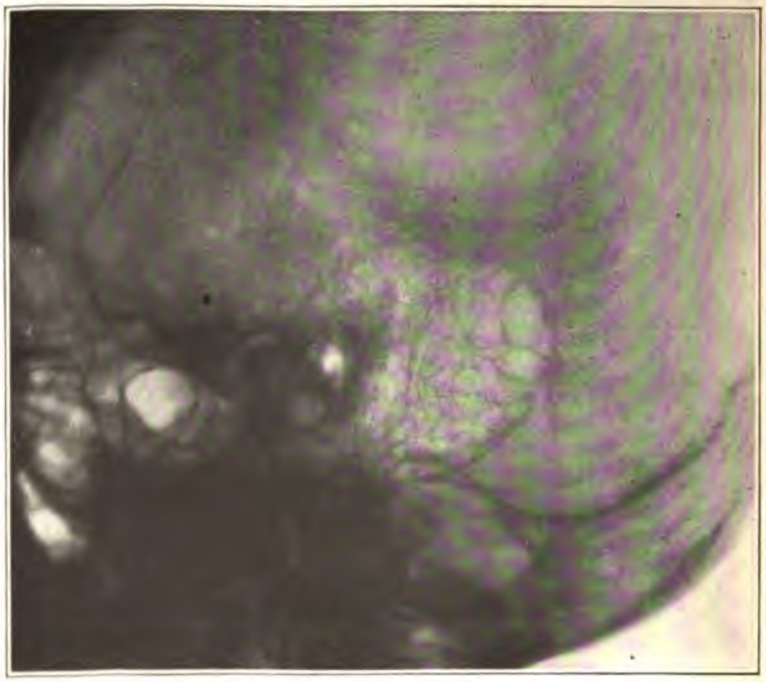


Fig. 82.—Mastoid cells normal. Large type.

The technic should be such that the frontal sinus, ethmoids, sphenoid sinus and antrum will be clearly defined.

We will not dwell upon dental diagnosis, even though it is of the utmost importance, as the student will find available several good works upon dental diagnosis. Several illustrations showing common pathologic dental findings are, however, hereto appended.

THE THORAX

The diagnosis of intrathoracic conditions should never be attempted from flat plates. The study of the normal picture is very necessary, as the appearance of the lung changes as the subject advances in age and also as the result of disease.

The air vesicles of the normal lung cast no shadows; but the lymphatics cast shadows which are more dense at the hilus and which decrease in distinctness toward the periphery. It is the various gradations in density from the normal to the pathologic lung which enable us to read the diagnosis into the plate. In a large number of cases the wrong opinion has been given in the reading of plates, especially in the case of pulmonary tuberculosis, owing to the lack of a clear interpretation of the shadow ties. This knowledge is only to be obtained through the of numerous plates under proficient guidance. There are shadows present upon the plate which have no relation

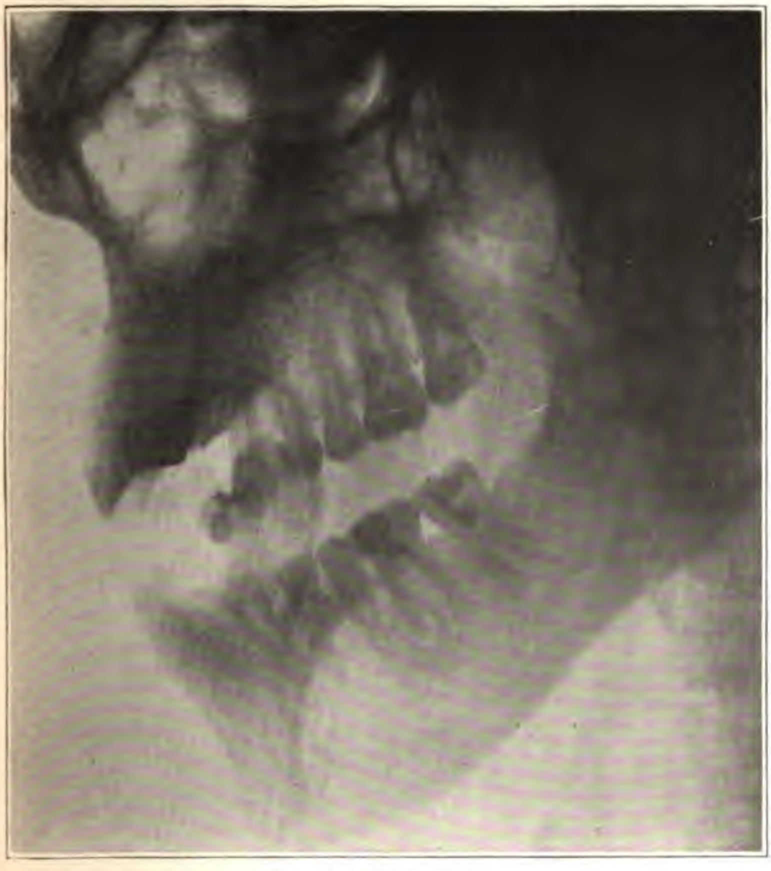


Fig. 83.—Apical abscess.

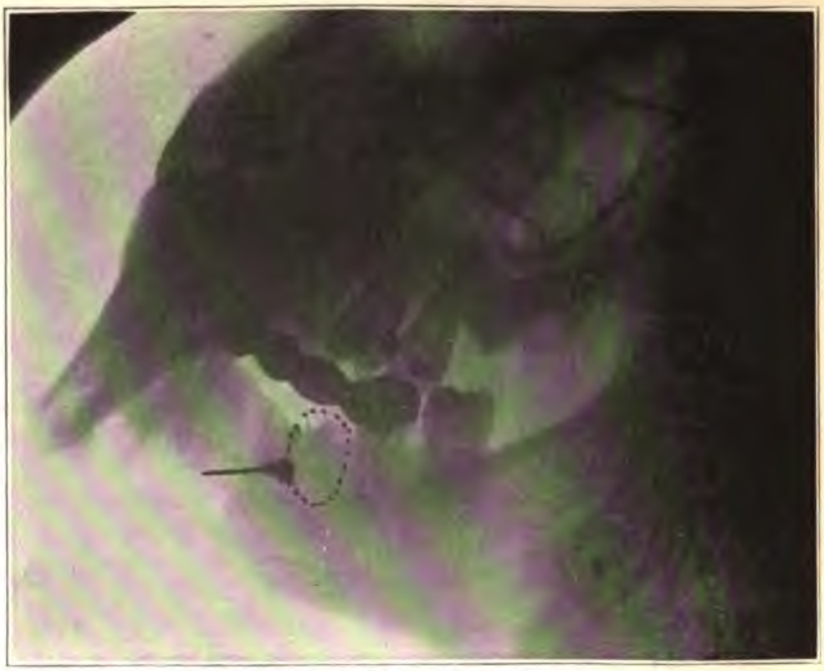


Fig. 84.—Old unextracted root.

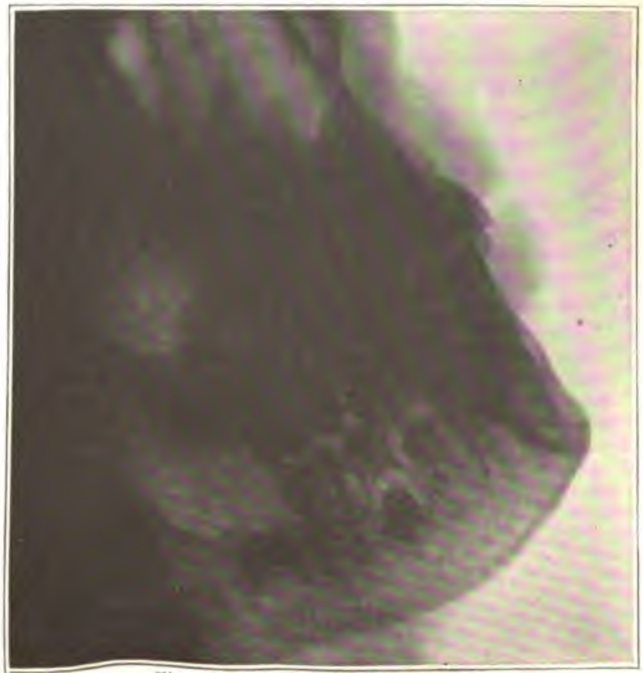


Fig. 85.—Unerupted teeth. Early life.

to the disease. Blood vessels, bronchi, calcareous areas, all
found in normal pulmonary tissue, and should not be mistaken
pathology. The breast of the female subject, the pectoral m
cles, and the scapulæ should be carefully noted in each case. S
ilarly, the hilus of the lung and the trachea cast shadows, wh
should be borne in mind when interpreting any plate.

The Diaphragm.—Radioscopy affords the most reliable me
of studying limitations and variations in the movements of
diaphragm. Upon fluoroscopic examination, variations in
movements, position, and general outline of this important m
cle may be noted. Unilateral limitation of the mobility of the d

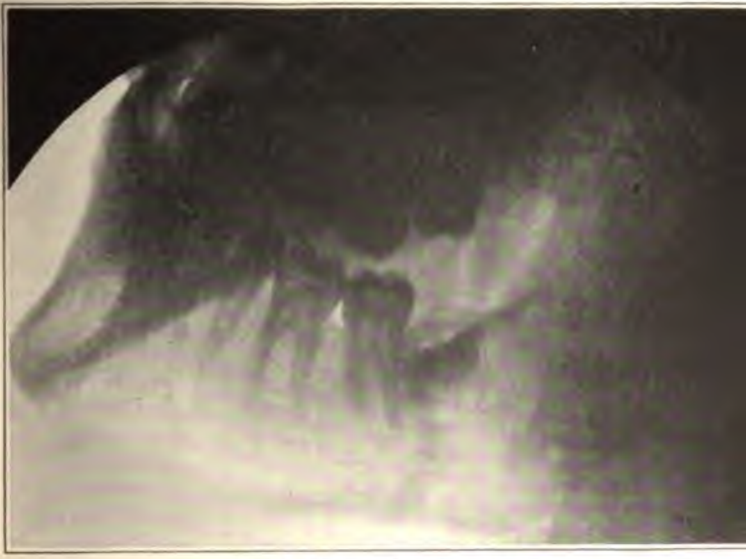


Fig. 86.—Unerupted molar. Adult.

phragm is often significant of incipient phthisis (Williams' sig
However, similar limitation of movement of the muscle may
caused by increased subphrenic pressure or by the traction
pleural adhesions.

The general outline of the diaphragm is altered in diaphr
matic paralysis and in the presence of diaphragmatic hernia.
the case of the latter affection straining or coughing will incre
the herniation of the sac contents.

Pulmonary Tuberculosis.—In tuberculosis of the lungs ra
oscopy of the thorax reveals multiple patches of mottling in
area involved, a diminution in the transparency of the pulmon
tissues, which is not, however, as dense or as sharply circu

scribed as is the shadow cast in lobar pneumonia. On the contrary, in pulmonary tuberculosis there are multiple areas of impaired transparency, often superimposed, and frequently not exceeding one-fourth inch in diameter. In incipient tuberculosis the mottling is usually circumscribed to the apical or axillary regions, the inferior portions of the lungs remaining clear at this stage of the disease.

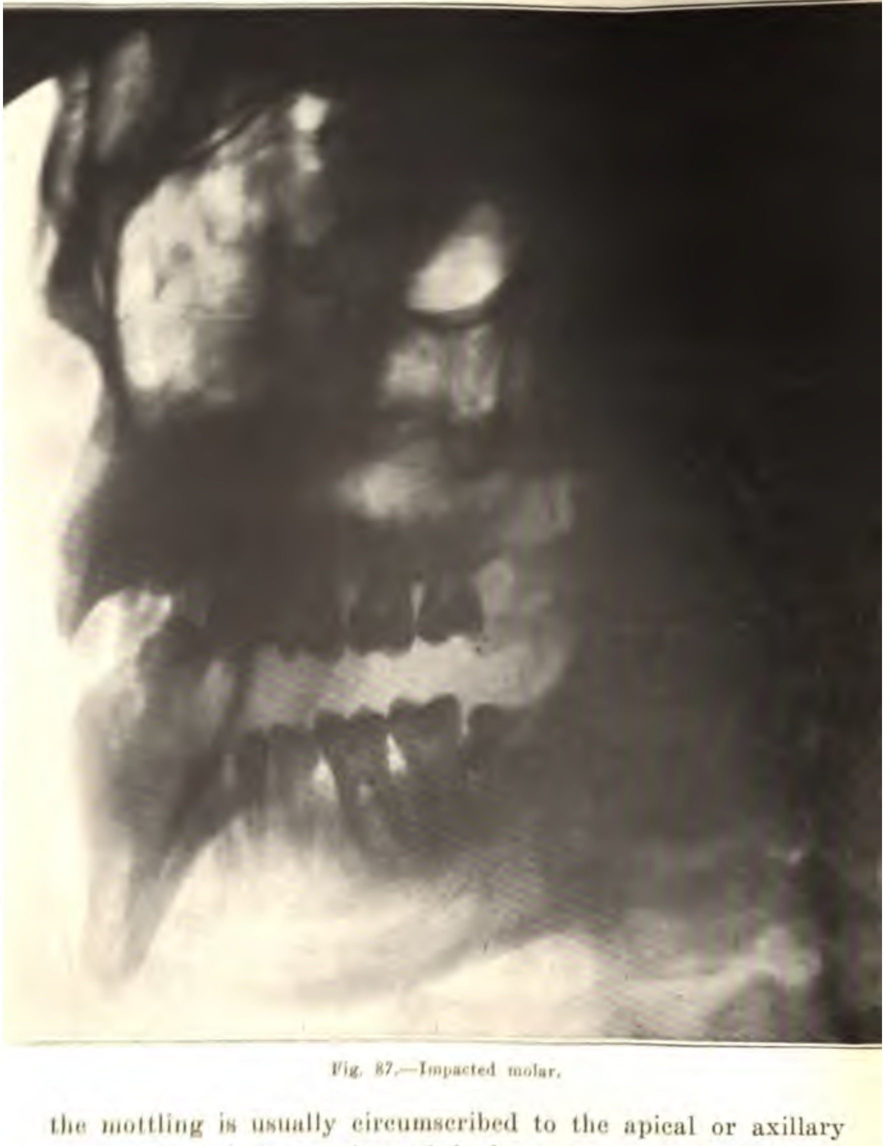


Fig. 87.—Impacted molar.

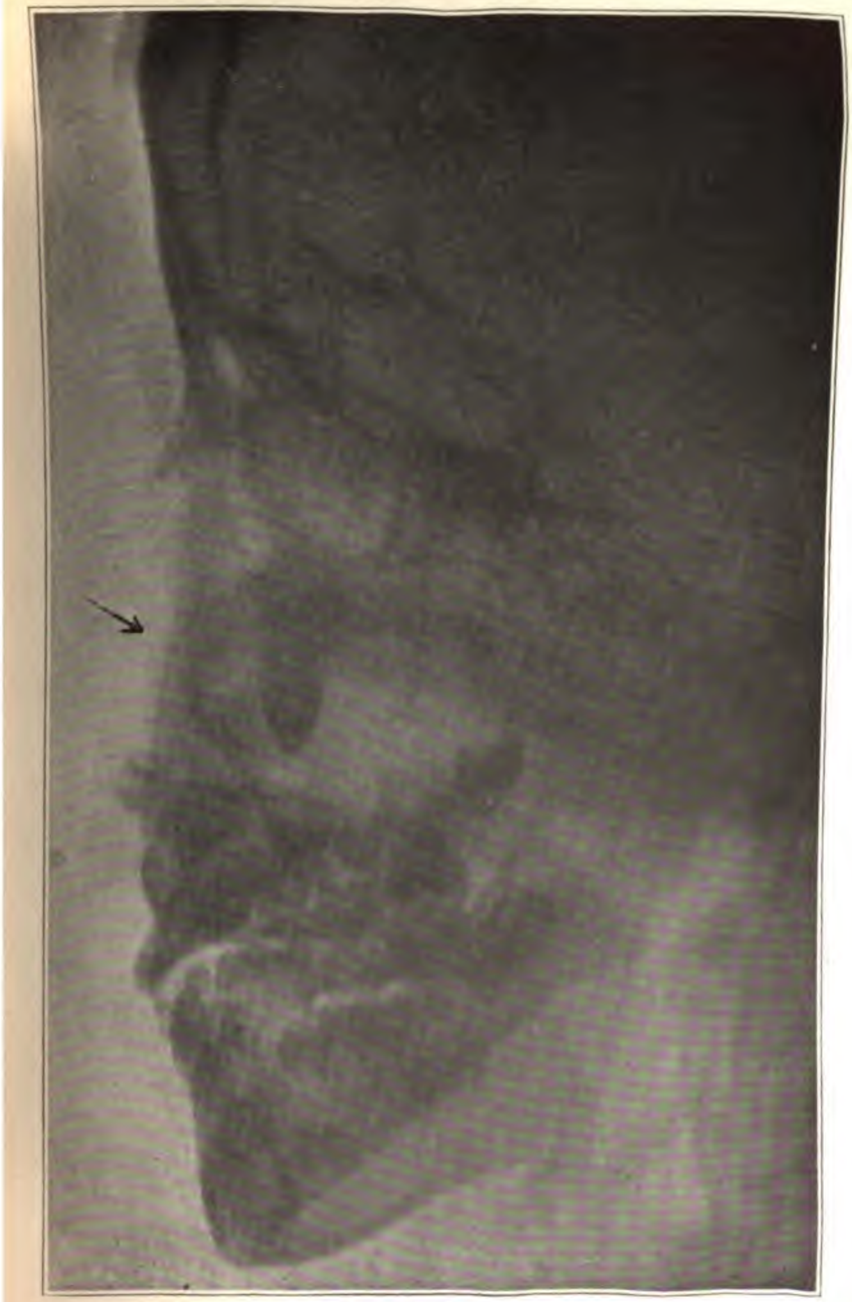


Fig. 88.—Unerupted canine.

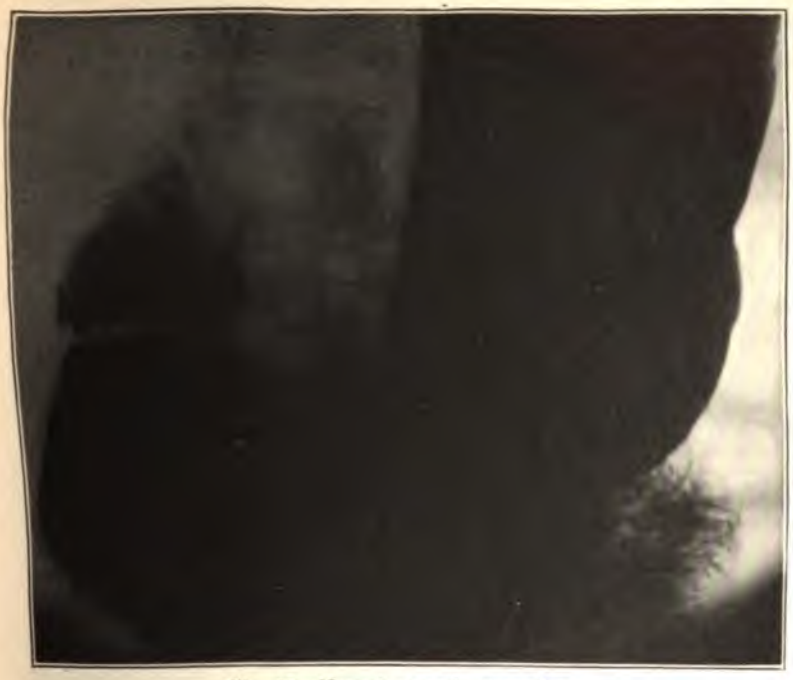


Fig. 90.—Normal stomach—normal cap.

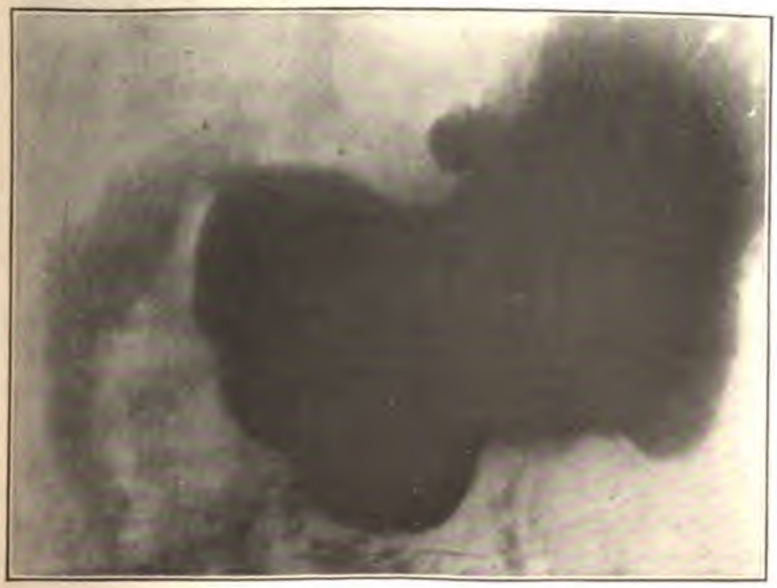


Fig. 91.—Penetrating ulcer of lesser curvature.

[illegible]

[illegible]

[illegible]

size, extent, and relation to surrounding structures may be studied. A solitary, large tumor casts a relatively dense shadow, whereas multiple small, metastatic growths show multiple patches of impaired transparency within a fairly small space in the upper portion of the mediastinum.

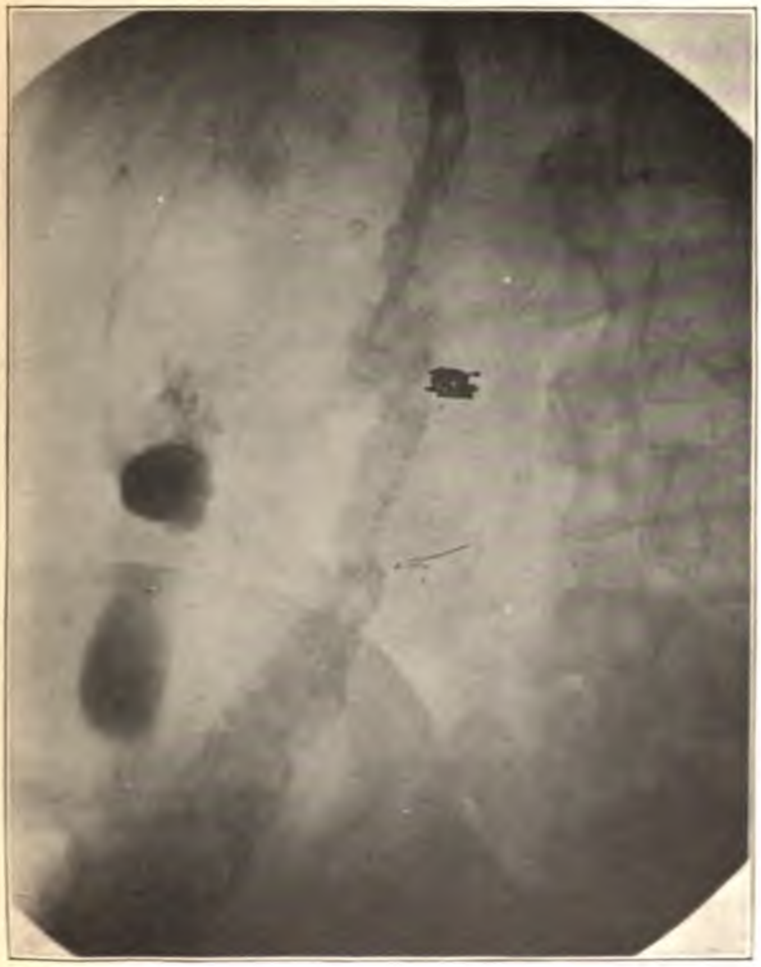


Fig. [illegible].—Deformed spine.

essary in order to render a clear-cut diagnosis. Cystouretero-pyeloroentgenography will aid materially in the diagnosis.

Pyelography.—The injection of certain substances which are opaque to the rays will demonstrate the renal pelvis and calyces, also the position of the ureters and bladder. Thorium nitrate, collargol, and argyrol have been used. At present we are using sodium iodide, which is found to be very satisfactory and non-irritating to the patient. It gives an excellent contrast on the plate.

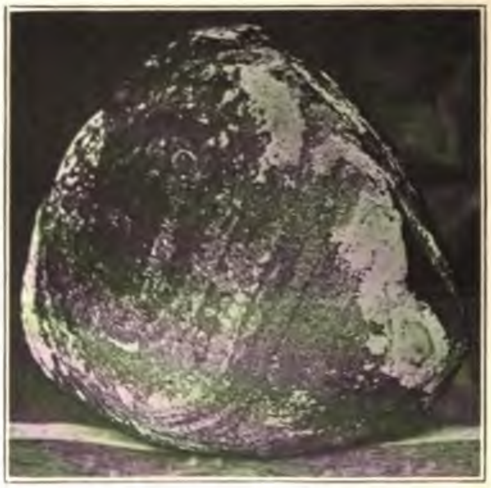


Fig. 98.—Calculus after removal.

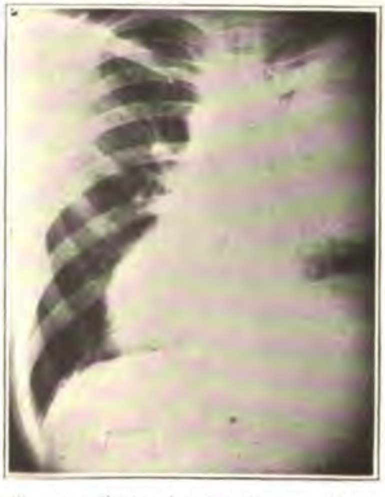


Fig. 99.—Encapsulated empyema. Right.

THE GASTROINTESTINAL TRACT

The examination of the gastrointestinal tract radiographicall is to a certain extent not fully understood by the average pra titioner. The technic of this examination radiographically i of paramount importance, provided the diagnosis is made by a experienced radiologist. The serial examination of the tract i very necessary to determine the presence or absence of pathologi

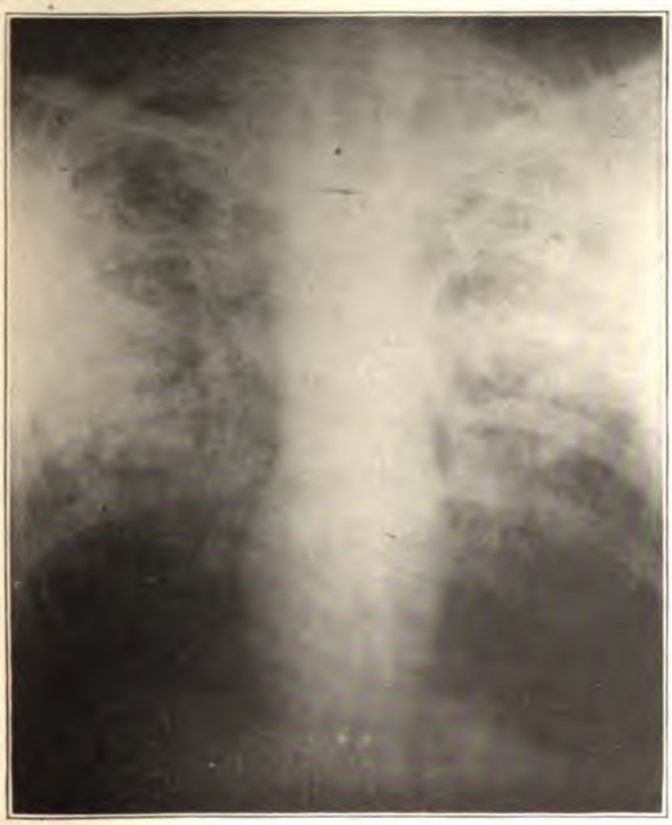


Fig. 100.—Tuberculosis of the chest with typical drop heart.

changes. The one or two plate method following a barium mea is to be condemned as a waste of the time of both physician an patient.

The findings of the gastrointestinal tract which we most com monly search for are gastric ulcer, duodenal ulcer, adhesion gall bladder disease, stones, and malignant disease. The spac allotted to this subject will not suffice for a discussion of th different diagnostic points; but I wish to insist upon the fact that a thorough study of the case should be made before a diagnosis is rendered.

The illustrations in this chapter it is hoped will afford the reader an adequate conception of the conditions which are best studied radiographically; and the perusal of a text together with the observation of a large number of plates will perfect the student in radiographic diagnosis.

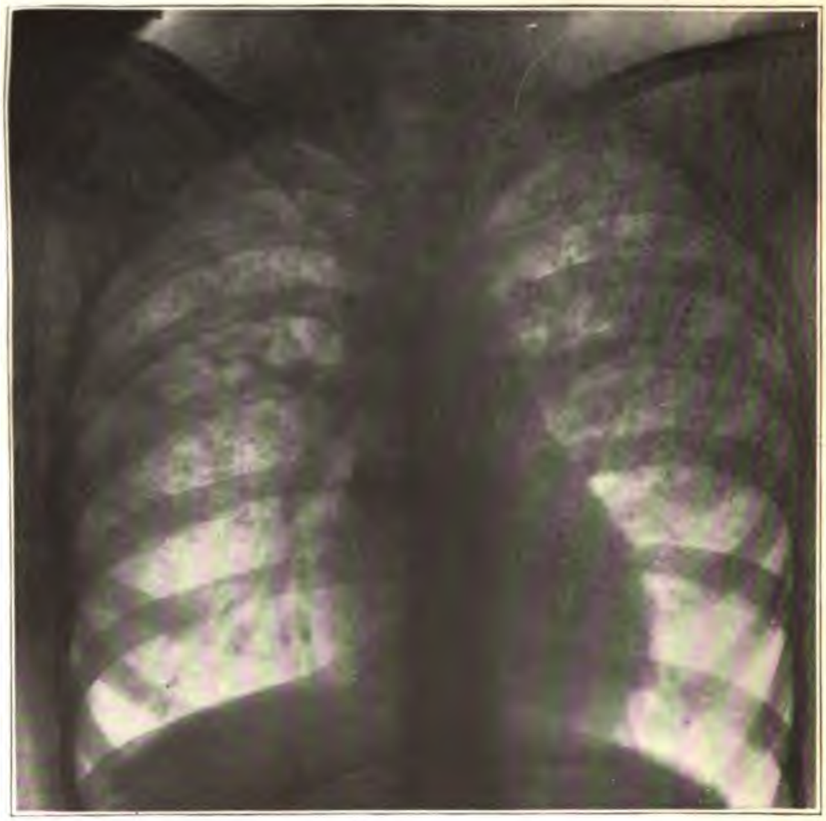


Fig. 101.—Tuberculosis of the lung.

SECTION III

DISEASES OF THE RESPIRATORY ORGANS

CHAPTER VIII

DISEASES OF THE BRONCHI

ACUTE BRONCHITIS

Clinical Pathology.—Acute bronchitis, an acute catarrhal inflammation of the mucous membrane of the medium-sized an larger bronchi, occurs as a primary affection, and as a complication of many of the acute infectious diseases, notably the exanthematous fevers, influenza, typhoid fever, and malaria.

The disease is most prevalent during the sudden changes o early spring and late autumn. Among predisposing causes ma be mentioned particularly acute coryza, affecting the upper respiratory passages; and passive congestion of the lungs inciden to regurgitant heart disease, acting upon the lower portions o the bronchial tree. The organism which is most frequentl causative is the pneumococcus, alone or in conjunction with th staphylococcus, the colon bacillus, the micrococcus catarrhalis or the bacillus typhosus.

During the early stages of the inflammation the mucous membrane of the bronchi is swollen and red, but is dry. During th further evolution of the disease, however, the congested mucou membrane becomes bathed with secretion, mucopurulent or purulent in character, containing large numbers of desquamated epithelial cells and bacteria.

Physical Signs.—Mild cases of acute bronchitis yield few physical signs which are characteristic of the affection. In the mor severe grades of bronchial inflammation physical signs are mor in evidence, but often require a very careful examination to afford definite diagnostic data.

Inspection in the milder cases usually reveals nothing abnormal, but in more severe cases there is moderate acceleration o he respirations; while if there is a complicating or concomitan ımmat [illegible] finer bronchioles (capillary bronchitis), th condition is attended by a considerable degree of dyspnea, and in the young subject, cyanosis.

Palpation, negative in cases of moderate severity, in well-developed cases of acute bronchitis may reveal slight rhonchal fremitus distributed over both lungs.

Percussion seldom elicits any alteration of the normal vesicular resonance in cases of frank acute bronchitis. Occasionally in very severe cases a slight impairment of resonance is evident over the bases posteriorly. The bases should be carefully percussed daily in order that a complicating bronchopneumonia may be recognized in its inception.

Auscultation during the early stage of the disease reveals the presence of sibilant and sonorous râles well distributed over both lungs. In a later stage of the affection, after the secretion has become freely established, moist râles appear, the crepitant râle predominating the clinical picture.

Vocal resonance is not perceptibly altered. The respiratory murmur is harsh or puerile; but in uncomplicated acute bronchitis the breath sounds are never purely bronchial.

Diagnosis.—The diagnosis of acute bronchitis rests upon the absence of physical signs other than puerile breathing and a few râles distributed over both sides of the thorax, coupled with certain subjective symptoms, as an initial chill or chilliness followed by moderate fever, a dry hacking cough which loosens with the establishment of the bronchial secretions; a feeling of rawness and pain beneath the sternum; and a general feeling of malaise and pain in the back and limbs.

Differential Diagnosis.—In its abrupt onset the disease frequently is suggestive of *lobar pneumonia;* but this disease is eliminated by the absence of physical signs of consolidation; namely, increased vocal fremitus and resonance, flatness, and blowing, tubular breath sounds. Moreover, lobar pneumonia is almost invariably a unilateral affection, whereas acute bronchitis is bilateral in its manifestations. The constitutional toxemia and depression of lobar pneumonia far surpass that which attends acute bronchitis.

Bronchopneumonia is usually gradual and insidious in its primary manifestations; and, in addition to the physical signs of a diffuse acute bronchitis, presents multiple areas of i
resonance, over which the respiratory sounds are br
the least are bronchovesicular. The constitut

accompanying bronchopneumonia is more pronounced, and th dyspnea is of a more extreme grade.

Pertussis, during the first week or ten days, cannot be differentiated from acute bronchitis; but after the development of th first "whoop" the diagnosis is readily made. A history of exposure to pertussis is often to be elicited.

CHRONIC BRONCHITIS

Clinical Pathology.—Chronic inflammation of the bronchial mucous membrane occurs secondarily to a great variety of bodil states. Adults and elderly persons are most frequently the subjects of chronic bronchitis, the disease constituting the regularl recurring "winter cough" of many persons past middle life.

A certain percentage of cases of chronic bronchitis develop a the result of frequently repeated attacks of acute bronchitis There is naturally great diversity of opinion as to the period a which an acute bronchitis shall be said to have become chronic Gintrac including in the latter group any case which persist longer than forty days.

In other instances the disease is secondary to circulatory disturbances in the lungs, developing as the result of valvular disease of the left heart, aneurysm of the thoracic aorta, genera arteriosclerosis, or renal disease. In this group should also b placed the cases developing so frequently in obese subjects.

Next in frequency to these causes of chronic bronchial inflammation comes chronic pulmonary disease. Hypertrophic emphysema is quite constantly attended by chronic bronchitis, as ar also chronic ulcerative phthisis, pneumonokoniosis, bronchiectasis, and bronchial asthma.

Certain constitutional diseases are causative of or are frequently attended by chronic bronchitis. This is notably true o gout and uremia, particularly in elderly subjects of these maladies. Rachitic subjects are very prone to develop chronic bronchial inflammation as are those of a strumous, scrofulous predisposition. Subjects of chronic eczema for some reason frequentl suffer from chronic bronchial symptoms, this group of cases con ing the "endormoses" of Gueneau de Mussy.

e continual exposure to irritating gases and dusts is of pneumonokoniosis and pulmonary fibrosis, so also ors in milder concentration causative of chronic cases constitute the "mechanical bronchitis" of Walshe. Similarly, constant inhalation of unwarmed air by mouth breathing due to nasal obstruction results in chronic catarrhal inflammation of the bronchial mucous membrane.

Chronic bronchitis occurs in a hypertrophic and in an atrophic form. In the former there is proliferation of the mucous or goblet cells of the mucous membrane, which pour out a viscid grayish secretion containing innumerable desquamated epithelial cells mixed with bacteria. The lumen of the bronchus is diminished in numerous areas by hypertrophic thickening of the mucous membrane. In the atrophic form, on the contrary, the mucosa in certain areas of the bronchial distribution is thinned; the muscularis is replaced by fibrous connective tissue; and the weakened bronchial wall shows fusiform or saccular ectases.

In addition to the simple catarrhal form of the disease, chronic bronchitis occurs in the form of several modified or special forms; notably as *purulent bronchitis*, the *catarrhe sec* of Laennec, *bronchorrhoea serosa* or *catarrhe pituiteux* of Laennec, as *fetid* or *putrid bronchitis*, and as *eosinophilic bronchitis*.

Purulent bronchitis, a form of extensive duration, is characterized by the expectoration of large quantities of purulent sputum of offensive odor, the foulness of the sputum, however, not approaching that of the putrid form of the disease. When the purulent expectoration is very abundant the term *bronchoblenorrhea* is applicable to the disease. These cases sometimes exhibit a febrile movement which may simulate chronic ulcerative phthisis; and in other instances the temperature is septic, with colliquative sweats, causing excessive discomfort to the patient, and occasionally actually endangering life.

The *catarrhe sec* of Laennec is a form of chronic bronchial inflammation which is attended by paroxysms of dyspnea and cough, simulating the picture of bronchial asthma, but with the distinguishing feature of very scanty expectoration. Cough is more pronounced toward the end of the attack, and with the establishment of free cough attended by expectoration the dyspnea is relieved to a moderate degree. The paroxysms of cough excite pain in the thorax which is usually circumscribed to definite limited areas of the chest wall corresponding to the attachment of the diaphragm.

The expectoration, which is absent during the initial dyspneic period of the disease, contains in addition to a few desquamated epithelial cells with leucocytes and bacteria, many small grayish pellets of mucus, the "crachats perles" of Laennec. Curschmann's spirals and Charcot-Leyden crystals are occasionally present in the sputum.

Believed by Laennec to occur only in subjects of gouty diathesis, this form of chronic bronchitis undoubtedly has a predilectio for those subjects; but it has been noted as an intercurrent condition in the course of other forms of chronic bronchitis and i cases of moderate pulmonary congestion, when this is not sufficient in degree to produce frank pulmonary edema. Catarrh sec is very prone to cause emphysema, and the two conditions ar not infrequently associated.

The bronchial mucous membrane in subjects of this disease i hypertrophied and acutely congested, leading to diminution o the bronchial lumen and predisposing to emphysema. Affectin in the main the smaller bronchi and the bronchioles, these are i certain instances completely occluded by the turgescence of th mucosa, constituting the "catarrhal bronchostenosis" of Cantani

In *bronchorrhea serosa*, or *catarrhe pituiteux* of Laennec, th pathologic antithesis of catarrhe sec is observed. This form o chronic bronchitis is characterized by the expectoration of larg amounts of fluid, which in cases of moderate duration is thin clear and frothy, containing small numbers of Curschmann's spirals and Charcot-Leyden crystals; but which in cases of extensiv duration is cloudy, mucopurulent, or finally purulent in character. The expectoration is very abundant, as much as a lite being raised in twenty-four hours in many instances. Cough i persistent, and, occurring in paroxysms, has led to the nam "asthma humidum" which is occasionally applied to the condition

The bronchial mucous membrane is atrophic, and the weight o the accumulating secretion predisposes to bronchiectasis, which i not infrequently present in the purulent form of the disease. Th purely serous form, true bronchorrhea serosa, has been attribute to nervous hypersecretion.

Fetid or ***putrid bronchitis*** is distinguished by the exceedingl foul odor of the expectoration, which contains characteristic small gray pellets, composed of mucus, with bacteria, pus cells, fatt acid crystals and cellular detritus, Dittrich's plugs. The sputu is yellowish-gray, abundant, and upon standing separates int three layers: an upper of yellowish froth, a middle of transparent clear fluid, and a lower of purulent sediment. In addition t putrid bronchitis, purulent expectoration occurs in bronchiec is, chronic ulcerative phthisis with cavitation, pulmonary ab ss and gangrene, and empyema with pulmonary fistula. Putri bronchitis is apt to produce aspiration pneumonia, pulmonary abscess, or gangrene of the lung, the patient exhibiting signs of sepsis, which are of grave prognostic import.

In the *eosinophilic bronchitis* of Teichmüller there are paroxysms of dyspnea which lead ultimately to emphysema of the apices and anterior borders of the lungs. The sputum is colorless and mucoid, containing many eosinophiles, desquamated epithelial cells and occasionally Curschmann's spirals and Charcot-Leyden crystals. Occurring in paroxysms, the disease is really to be considered a rudimentary form of bronchial asthma in which the chronic catarrhal symptoms predominate the clinical picture.

The ultimate result of persistent chronic bronchitis is pulmonary emphysema. The partial occlusion of the bronchioles combined with the paroxysms of violent cough are the prime factors in the production of this condition. However, the chronic bronchial irritation also results in peribronchial fibrosis, which by favoring interstitial sclerosis of the lung, also predisposes to bronchiectasis from traction. In other instances signs of right heart incompetence are manifested, as the result of interference with the pulmonary circulation, and in the absence of emphysema there is evidence of chronic venous stasis.

Physical Signs.—*Inspection.*—The subject of chronic bronchitis is liable to chronic shortness of breath, often attaining to the degree of actual dyspnea, upon moderate exertion. The paroxysms of violent cough in certain cases are suggestive of bronchial asthma. In the cases in which emphysema is coexistent the barrel chest of this disease with its limitation of expansion is noted, while in the circulatory group of cases there is apt to be cyanosis and edema from venous stasis.

Palpation.—Usually negative, palpation in cases associated with abundant secretion may reveal the presence of rhonchal fremitus of bilateral distribution. Vocal fremitus is little altered if indeed at all.

Percussion.—The percussion note in chronic bronchitis is often quite normal. However, in cases attended by emphysema the note is hyperresonant; whereas in cases of fetid bronchitis associated with bronchiectasis, dullness is encountered over large superficially filled ectases, the note changing to hyperresonance or tympany upon the expulsion of the contents of the cavities. Similarly there is in cases of cardiac origin not infrequently impairment of resonance over the bases posteriorly as the result of moderate pulmonary edema.

Auscultation.—Auscultatory signs in chronic bronchitis are abundant and very variable. In chronic bronchitis of extensive duration the respiratory murmur is harsh and by reason of the accompanying emphysema expiration is prolonged. Râles of all types are audible over both sides of the thorax, particularly over the bases posteriorly, at the inferior angle of the scapula, and anteriorly in the infraclavicular and mammary regions. In simple chronic catarrhal bronchitis large and small râles are in evidence as is also the case in bronchorrhea serosa, associated with bronchiectasis. In catarrhe sec, on the contrary, the râles are dry, high pitched, and sibilant, moist râles being present in but small number if indeed at all.

Auscultation of the heart in chronic bronchitis yields variable and suggestive signs in many instances. In simple catarrhal bronchitis of comparatively brief duration the cardiac tones are unaltered; whereas in cases of cardiac origin and in cases which are associated with well-established emphysema, the second sound of the heart at the pulmonic area is accentuated; and in cases which are dependent upon chronic valvular disease the murmurs of the provocative lesions are audible. In cases of renal origin, on the contrary, the second sound at the aortic area is very frequently accentuated.

Diagnosis.—With a history of chronic cough recurring every winter, with râles distributed universally throughout both lungs, unattended by fever or loss of weight, in an elderly person who presents a thorax approaching the emphysematous type, the diagnosis of chronic catarrhal bronchitis is suggested. It should be borne in mind, however, that aortic aneurysm or mediastinal tumor are occasionally productive of chronic cough; but in these cases there is apt to be more or less pronounced stridor owing to unilateral vocal cord paralysis.

Catarrhe pituiteux, with its abundant, serous frothy expectoration, is apt to be confused with pulmonary edema; whereas putrid bronchitis with its foul sputum is apt to simulate the clinical picture of bronchiectasis. In most cases a differential diagnosis between putrid bronchitis and bronchiectasis with decomposition of the contents of the ectases may only be made when the latter disease forms cavities of sufficient size to give distinctive and definite physical signs.

In dealing with a disease which is so frequently secondary to other pathologic changes in the thorax, the diagnosis cannot be sidered complete until the causative lesion is in each case determined. In cases of cardiac origin auscultation of the precordia frequently reveals the presence of regurgitant lesions or signs of myocardial degeneration; while in cases dependent upon chronic renal disease the aortic second sound is accentuated and frequently there are signs of left ventricular hypertrophy. Again in cases of putrid bronchitis associated with pulmonary abscess or gangrene, careful physical examination will elicit confirmatory signs of the conditions. The eosinophilic form of chronic bronchitis is differentiated from bronchial asthma by the history in the latter disease of attacks of expiratory dyspnea and orthopnea dating from adolescence or early life, with intervals during which the patient is entirely free from pulmonary symptoms. The blood picture shows a higher degree of eosinophilia in true bronchial asthma.

It is of the utmost importance in cases of long standing chronic bronchitis to exclude the presence of chronic ulcerative phthisis, as the former is often part and parcel of the latter. In such event a careful search should be made for disease of the heart, arteries, or kidneys which might be responsible for the chronic bronchial inflammation. It should be recalled that a nontuberculous chronic bronchitis is not attended by febrile elevation, marked anemia, or loss of weight. Moreover, the physical signs in chronic bronchitis are bilateral, whereas in phthisis they are apt to be unilateral and to manifest a predilection for the pulmonary apices. In purulent and putrid bronchitis the sputum contains characteristic bodies in the form of Dittrich's plugs, Curschmann's spirals, and Charcot-Leyden crystals, whereas tissue shreds and elastic fibers, signs of tuberculous excavation of the lung, and the tubercle bacillus are absent from the sputum.

FIBRINOUS BRONCHITIS

Clinical Pathology.—Fibrinous coagula are formed in the smaller bronchial tubes under a variety of conditions. They have been noted in chronic disease of the lungs and heart; as a result of the inhalation of irritative gases, notably ammonia gas; as the result of infection of the bronchial mucous membrane by the pneumococcus or the Klebs-Loeffler bacillus, in the last instance the morbid process constituting a bronchial diphtheria. Following hemoptysis fibrinous masses are not infrequently expectorated, which may simulate those of fibrinous bronchitis.

Aside from these conditions, there are two conditions which may be classed as true fibrinous bronchitis with the expectoration of fibrinous moulds of the bronchial tubes amid paroxysms of dyspnea and cough. This essential fibrinous bronchitis occurs in two forms; namely, as *acute fibrinous bronchitis*, and as *chronic idiopathic fibrinous bronchitis.* The acute form of the disease is occasionally noted as a complication of the acute infectious fevers, notably in the course of scarlatina, measles, pneumonia, influenza, variola, tuberculosis, and typhoid fever. Fifteen cases of this type of the disease are reported by Bettman. Chronic idiopathic fibrinous bronchitis occurs as a chronic, recurrent malady, and the disease invariably attacks the same portion of the bronchial tree. In Bettman's collection of cases ten were accompanied by organic cardiac disease; fourteen developed during the course of pulmonary tuberculosis; five cases were associated with bronchial asthma; and four cases were attended by edema of the lungs.

The distinguishing feature of the disease is the expulsion from the bronchi and bronchioles of branching, fibrinous casts with the expectoration at the conclusion of a paroxysm of dyspnea and cough, which is relieved by the expulsion of the casts. The casts, which correspond in shape and size to the ramifications of the portion of the bronchial system which lodged them, vary in length and have been found to exceed ten centimeters in length. The body of the cast, which is composed largely of superimposed laminæ of fibrin with epithelial cells, leukocytes, bacteria, cellular detritus, and occasionally Charcot-Leydon crystals, corresponds in diameter with the lumen of the bronchial tube from which it was expelled.

Much confusion still remains as to the exact nature of the pathologic changes which occur in the mucous membrane of the bronchi in the affected areas. Weigert and Kretschy reasoned that the epithelial surface must desquamate in order to render possible the formation of the casts. Bettman, quoting Schittenhelm, describes a desquamative catarrhal inflammation of the alveoli with exudation into the alveoli, bronchioles, and smaller bronchioles.

Physical Signs.—*Inspection.*—During the paroxysms of dyspnea d cough which characterize the attacks the patient is usually pneic, the clinical picture simulating that of the paroxysm of ial asthma. The dyspnea is often relieved by the sudden copious expectoration, while in other instances the attack is attended by distressing cough with very scanty expectoration.

Palpation.—As a rule, palpation yields but limited data in this disease. However, when one of the larger bronchi is occluded by a cast, vocal fremitus is absent over the distribution of its radicles, and the expansion of the corresponding side of the thorax is diminished.

Percussion.—In the absence of concomitant disease of the lung, percussion of the thorax is usually negative in cases of fibrinous bronchitis. However, when a large bronchus is occluded the note is impaired over the area of the thorax corresponding to its distribution, to become resonant once more upon the expulsion of the offending cast. Moreover, it is occasionally possible to elicit a moderately hyperresonant note over the adjacent pulmonary tissues in the presence of bronchial occlusion, as a result of compensatory emphysema of the neighboring vesicles.

Auscultation.—Moist râles are frequently encountered, while occasionally the bruit de drapeau is audible. When present, it is of considerable diagnostic value. The respiratory murmur is absent over the distribution of an occluded bronchus, only to become reestablished with the expulsion of the cast and the restoration of the patency of the tube. The intensity of vocal resonance undergoes similar modifications under the same conditions.

Diagnosis.—Fibrinous bronchitis with its attacks of paroxysmal dyspnea may readily be confounded with bronchial asthma. The ultimate differential diagnosis depends upon the recognition of typical fibrinous casts in the sputum. While the physical signs are rarely typical of the disease, the presence of the bruit de drapeau is of great aid in diagnosis; and Andral has called attention to the importance of the transient abolition of the respiratory murmur over a circumscribed area of the thorax combined with normal pulmonary resonance. In all cases the presence of bronchial diphtheria and hemoptysis must be excluded.

BRONCHIECTASIS

Clinical Pathology.—Bronchiectasis is in the vast majority of cases a secondary disease, complicating previously existing disease of the bronchi, the lungs, or the pleura.

The dilatation may be produced by increased pressure exerted upon a weakened bronchial wall from within, or by traction exerted upon the bronchial wall from without. In chronic bronchitis of long standing, with its paroxysms of violent cough, combined with the accumulation of the abundant secretion in the purulent and putrid forms of the disease, the weakened bronchial wall is apt to yield and lead to bronchiectasis. The violent paroxysmal cough of pertussis may result similarly. Bronchial dilatation follows stenosis of the bronchi occurring as a result of syphilitic or tuberculous ulceration, and it has developed as a result of bronchial compression by mediastinal tumor or aneurysm.

The traction of adhesions upon the bronchial wall in chronic interstitial pneumonia, pulmonary syphilis, and fibroid phthisis is one of the most productive etiologic factors in the production of bronchiectasis. In these cases the cicatricial bands may pass from a thickened pleura to the affected bronchus, or they may pass from one bronchus to another. Bronchiectasis has followed the lodgment of foreign bodies in the bronchi. In these cases Cohn holds that the dilatation develops at the site of the foreign body as a result of irritation and consequent ulceration, and not distal to it as a result of stenosis. Bronchiectasis occasionally develops in connection with lobar or lobular pneumonia, chronic ulcerative phthisis, and pulmonary neoplasm. Moreover, bronchiectasis occurs in a congenital form, the "bronchiectasis universalis" of Grawitz.

Bronchiectasis is not a common disease. The cases have usually developed in young adults and in persons of middle age; and males have been affected with the disease more frequently than have females.

The bronchial dilatations are found most frequently in the right lung, in which they affect principally the bronchi of the middle and lower lobes. The dimensions of the ectases are variable, ranging from a moderate increase in the lumen of the tube to large cavities in which the bronchial communication is often obliterated. Two principal forms of dilatation are encountered; namely, the *saccular* and the *cylindrical*. Cylindrical dilatation is most frequently observed in connection with the smaller bronchial tubes; but it is also encountered in the larger bronchi; and the two forms, saccular and cylindrical, are not infrequently found in the same lung.

The pulmonary tissue adjacent to the larger ectases presents reas of compensatory emphysema, alternating with areas of sclero-n which the pleura often participates. The number and dis-on of the bronchiectatic areas are always variable; but as a rule a single large bronchiectasis occupies a position rather deep within the interior of the lung, whereas multiple small bronchiectases show a more superficial distribution.

The state of the mucous membrane of the dilatations varies. In certain instances scarcely altered, in practically all extensive dilatations it is thickened, with polypoid elevations upon the surface; while in cavities containing abundant secretion the mucosa is not infrequently ulcerated. The exudation from the walls of extensive bronchiectatic dilatations is usually purulent and abundant; but occasionally it is scanty and cheesy in consistence.

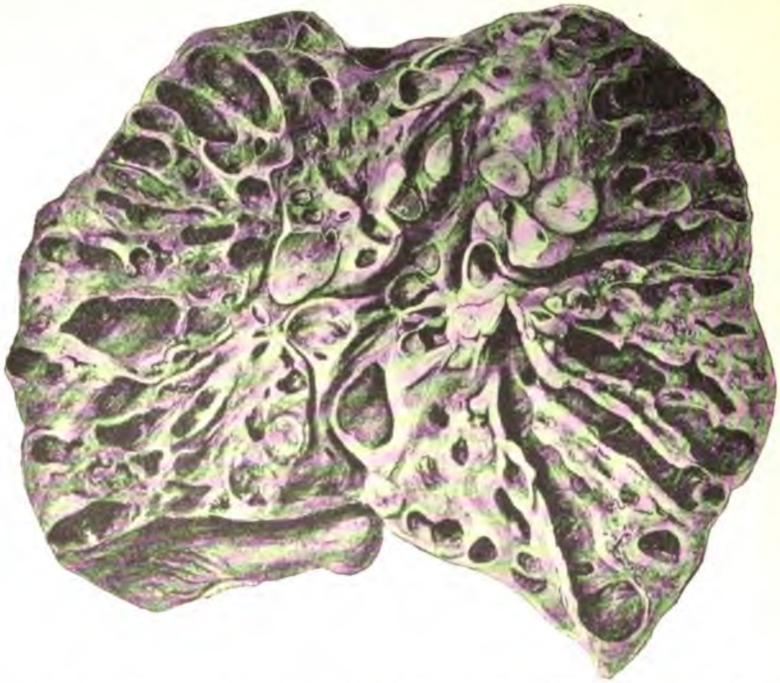


Fig. 102.—Sacculated bronchiectasis. (Pottenger, after Powell and Hartley.)

The expectoration, which represents the contents of the ectases, is gray or brown in color, mucopurulent in character, and in cases with large cavities is abundant and fetid. Upon standing, the sputum separates into three strata; an upper of brownish froth, a middle of semiopaque fluid, and a lower composed of granular sediment containing cellular detritus, bacteria, fatty acid crystals, leucocytes, and occasionally Charcot-Leyden crystals. The disease is occasionally, though rarely, attended by hemoptysis.

Bronchiectasis not infrequently results in emphysema, and these cases, constituting the "dry cases" of the disease, give the most favorable prognosis. In cases with large ectases and cavitation, associated with the collection of abundant secretion, abscess or gangrene of the lung is apt to supervene. Abscess of the brain has been noted in connection with bronchiectasis, usually upon the side corresponding to the site of the dilatation.

Physical Signs.—*Inspection.*—The presence of small bronchiectases produces no characteristic physical signs. In the fully established case of bronchiectasis, however, in which the dilatations have attained considerable size, there is impairment of expansion of the corresponding side of the thorax and occasionally retraction of the affected side with drooping of the shoulder. The decubitus of the patient, while variable, in advanced cases is suggestive; as the patient usually prefers to lie upon the affected side, and a change of posture is frequently followed by a severe paroxysm of cough due to shifting of the contents of the cavities.

Fluoroscopy in advanced cases is apt to reveal a shadow in the area of an extensive dilatation which contains mucopurulent material, this area clearing perceptibly upon evacuation of the contents during a paroxysm of cough. Clubbing of the finger-tips from enlargement of the terminal phalanges is present in many cases of long standing.

Palpation.—In the presence of a large bronchiectatic cavity with patent bronchial communication, situated superficially in the thorax, vocal fremitus is very markedly increased when the cavity is empty, to become abolished over the same area when the cavity is filled with fluid.

Percussion.—The findings upon percussion in bronchiectasis are variable, depending upon whether the cavity under investigation is empty or is filled with secretion. If the cavity contains fluid, even though it has a patent bronchial outlet, the percussion note is flat; whereas, if the cavity is empty, it yields tympany or a cracked-pot sound upon percussion. In suitably situated cavities all of the signs of pulmonary cavity, such as Wintrich's change of sound, Friedreich's respiratory change of sound, and the change of sound of Gerhardt, may be elicited.

In every case in which the signs of cavitation are evanescent, present and absent at successive examinations, the possibility of nchiectasis should be borne in mind, as these cavities fill with etion which masks all physical signs, and then the signs of cavitation reappear with the evacuation of the contents of the dilatations.

Auscultation.—In cases of bronchiectasis which are of relatively short duration, auscultation yields only the signs of chronic bronchitis, puerile breath sounds, and râles. If, however, a rather large dilatation be properly situated with reference to its bronchial communication, amphoric breathing is encountered. If a large dilatation is situated near the periphery of the lung, and if it has a free bronchial outlet, vocal resonance is very greatly exaggerated, perhaps to the extent of affording bronchophony or whispering pectoriloquy.

Diagnosis.—Cases of moderate bronchiectasis are distinguished from chronic bronchitis with difficulty, of which disease indeed it is often a sequela. In well-established cases, however, in which more extensive organic change has occurred in the bronchial system, the expectoration of a copious amount of mucopurulent sputum at one time, followed by an absence of expectoration for several hours, is suggestive of bronchiectasis; and when in addition signs of cavitation can be elicited over the base of the lung the presence of bronchiectasis may be assumed.

Differential Diagnosis.—The most important point in differential diagnosis is the possibility of mistaking bronchiectasis for chronic ulcerative phthisis with cavity formation. However, vomicae in the latter disease are usually situated in the pulmonary apex, a region of the lung which is rarely invaded by bronchiectasis. Moreover, percussion of the lung adjacent to the cavity in bronchiectasis is apt to yield hyperresonance due to compensatory emphysema, whereas in phthisis similar percussion elicits dullness of consolidation, which at this advanced stage of the disease is usually demonstrable also upon percussion of the opposite lung. In phthisis the sputum is raised at frequent intervals and is apt to contain the tubercle bacillus and elastic fibers. Moreover, chronic ulcerative phthisis produces characteristic deformity of the thorax with fever, anemia, and night sweats, while the course of the disease is progressively downward to a fatal termination. In bronchiectasis, on the contrary, unless it develops upon a tuberculous basis, the physical signs persist for a long period, while the patient remains in a comparatively good state of health.

Empyema with rupture into a bronchus is attended by the sudden expectoration of a large amount of purulent sputum; but instead of signs of cavity over the lower lung, there is the dullness of the original empyema, with the probable presence of Grocco's sign.

Pulmonary abscess and gangrene, which may simulate bronchiectasis, are excluded by their more rapid evolution, and by the presence of signs of sepsis. Putrid bronchitis is excluded by its bilateral distribution.

BRONCHIAL ASTHMA

Clinical Pathology.—Bronchial or spasmodic asthma consists essentially of a paroxysmal dyspnea which is almost entirely expiratory in type, the subject of the disease during the paroxysm being unable adequately to expel the tidal air from the lungs. Bronchial asthma has nothing in common with the so-called cardiac asthma or renal asthma.

Numerous theories have been advanced in the effort to explain the cause of the asthmatic paroxysm. It has been suggested that the attack is initiated by a sudden spasm of the circularly disposed bronchial musculature; and that the obstacle to the egress of the tidal air from the lungs is due to narrowing of the lumen of the bronchioles by temporary turgescence of the mucous membrane. Curschmann held that the underlying cause is to be found in inflammation of the smaller bronchioles, the so-called *bronchiolitis exudativa* of this author. Spasm of the diaphragm has likewise been advanced as a probable cause of the paroxysm.

Recent clinical work would seem to indicate that a certain proportion of asthmatics are hypersensitive to some form of protein, and that the asthmatic paroxysm is to be attributed to this cause.

In the cases which have been subjected to autopsy the ciliated epithelium has been found in a state of desquamation, with bronchial congestion and exudation rich in eosinophilic cells.

The circulating blood during the paroxysm of bronchial asthma contains an excess of eosinophiles, these cells representing 25 per cent to 35 per cent of all the leucocytes.

The sputum in bronchial asthma is characteristic of the disease. In the early stages it is scanty and very tenacious, containing Curschmann's spirals and Charcot-Leyden crystals. Macroscopically Curschmann's spirals are white or yellow, assuming the form of twisted threads or of small balls. The length of the spiral rarely exceeds half an inch, but it may exceed two inches certain instances. Under the microscope they appear as mu-

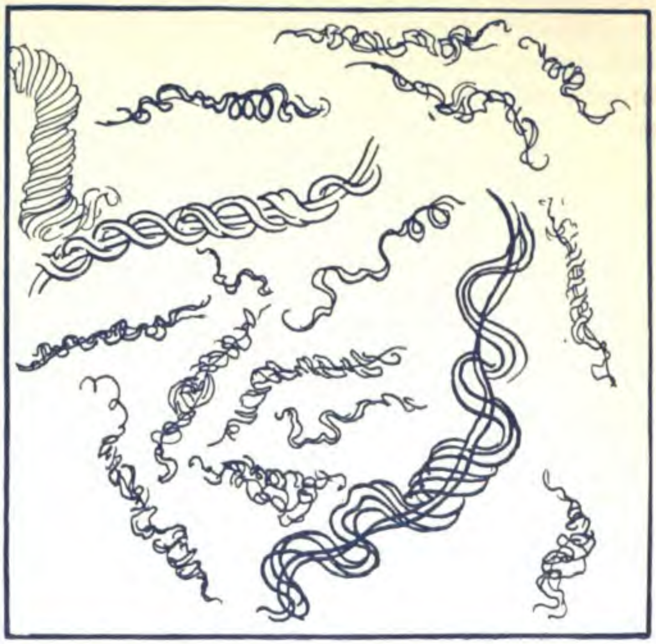
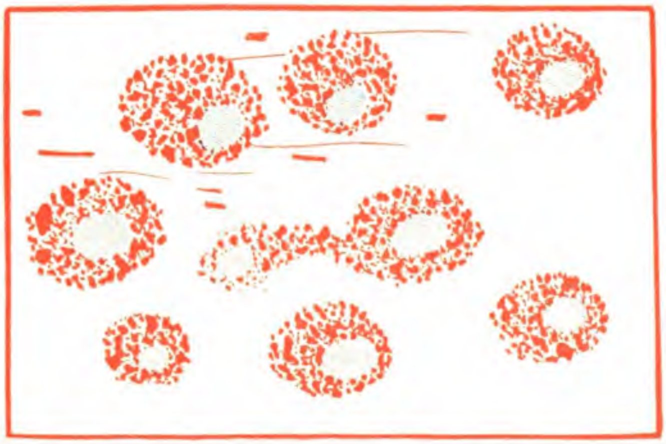


Fig. 103.—Curschmann's spirals. (From Brown.)



ig. 104.—Eosinophiles. A considerable percentage of the pus cells of asth
im are eosinophiles. This is probably indicative of chronic intoxication. (
vn.)

cous threads containing a clear, central fiber around which are wound numerous fine fibrils. Charcot-Leyden crystals occur as colorless, pointed, octohedral crystals, the average length of which is about three times the diameter of a red blood cell. They are frequently absent from the freshly expectorated sputum, but they appear after it has stood for a short time.

During the later stages of the attack these two pathognomonic elements of the sputum disappear, the expectoration becoming more abundant and mucopurulent in character.

Chronic bronchitis is frequently a concomitant affection in subjects of bronchial asthma; and the repeated paroxysms of the disease, with their typical expiratory dyspnea, tend to the production of emphysema and bronchiectasis.

Physical Signs.—*Inspection.*—During the paroxysm of bronchial asthma, which is very abrupt in onset, dyspnea of the expiratory type dominates the other physical signs. There is little if any increase in the number of respirations; and indeed, owing to the great prolongation of expiration, the number may be actually diminished. Inspiration is short and powerful, but adds little to the degree of expansion of the thorax, which remains fixed in a position of relatively full inspiration. Bamberger describes the inspiratory phase as that of normal inspiration with the participation of the accessory muscles of respiration in the act. Expiration, which lasts three to four times as long as inspiration, follows the latter promptly without the intervention of the normal pause between these phases, the accessory muscles of expiration being called into play in the attempt to empty the thorax.

During the course of the paroxysm the patient becomes orthopneic; the cervical veins become engorged; and the lips, face, and hands are apt to become cyanotic. The thorax is large and semifixed in the position of inspiration; the diaphragm is depressed; and the moderate degree of expansion during inspiration is exerted largely in the vertical direction, the thorax rising and falling *en masse* much in the same manner as in hypertrophic emphysema.

Palpation.—During the paroxysm vocal fremitus, if its intensity can be determined, is diminished as a result of the rarefaction of the transient acute vesicular emphysema or of bronchial obstruction by turgescence of the bronchial mucosa or of spasm of the bronchial muscles. Rhonchal fremitus is present to a striking ree, and is bilateral in its distribution. The cardiac impulse is masked by the concomitant emphysema of the anterior pulmonary borders. Pulsation is frequent in the cervical vessels and in the episternal notch.

Percussion.—The quality of the percussion note in bronchial asthma varies from a moderate hyperresonance during mild attacks to a note which closely approximates tympany in severe attacks in emphysematous subjects. The limits of pulmonary resonance are extended in all directions, upward in the supraclavicular regions, downward encroaching upon the hepatic and splenic dullness, and anteriorly, causing diminution in the area of cardiac dullness. In advanced cases, which are associated with marked emphysema, the area of cardiac dullness is frequently displaced downward. The respiratory excursion of the pulmonary borders is strikingly diminished in these subjects.

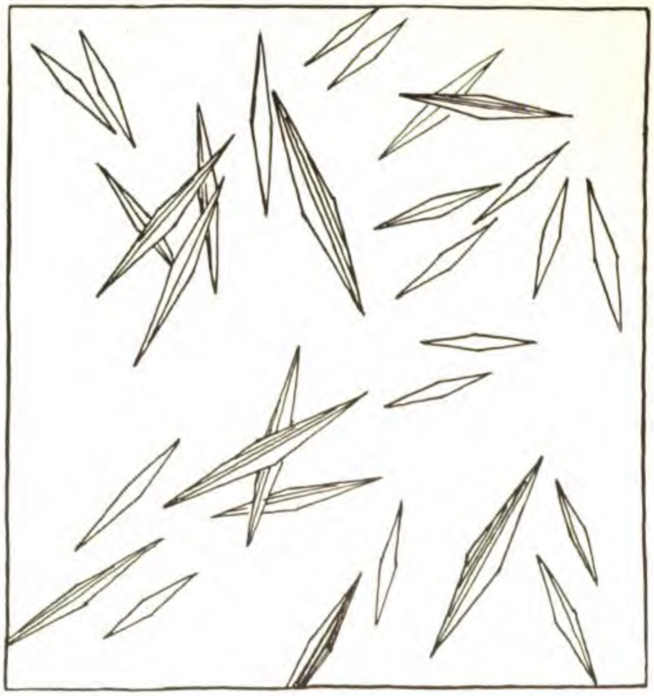


Fig. 105.—Charcot-Leyden crystals. These crystals are formed in sputum of chronic bronchitis, especially if asthma exists. They have been repeatedly found in other locations. They seem to indicate decomposition. (From Brown.)

Auscultation.—The vesicular murmur is obscured by numerous loud, sibilant and sonorous râles, which are so pronounced as frequently to be audible at some distance from the patient. Late in the paroxysm, after the bronchial secretion has become freely established, the dry râles give place to numerous moist and bubbling râles. Inspiration is frequently entirely inaudible, whereas the expiratory phase of the vesicular murmur is greatly prolonged and is punctuated with numerous râles.

Diagnosis.—Owing to the paroxysmal character of the asthmatic attack, with its well-marked expiratory dyspnea dotted with râles, the overdistention and relative fixation of the thorax, and the characteristic elements of the sputum, a diagnosis of bronchial asthma is often readily made. However, it is not infrequently necessary to differentiate the disease from attacks of dyspnea arising from other causes. Prominent among the latter is dyspnea due to laryngeal obstruction from spasm or edema of the glottis. In this case the dyspnea is distinctly of the inspiratory type, often with stridor, and as a rule is attended by marked respiratory movement of the larynx. There is often aphonia; the thorax is of normal size; and there is apt to be inspiratory recession of the lower intercostal spaces. The absence of râles in the presence of laryngeal obstruction is in marked contrast to the numerous râles of the asthmatic attack.

Differential Diagnosis.—Bronchial compression by enlarged glands, tumors, or aneurysm, and stenosis resulting from the ingestion of foreign bodies are to be excluded before a diagnosis of bronchial asthma is to be made upon the presence of paroxysms of severe dyspnea.

Hypertrophic emphysema, chronic bronchitis, and bronchial asthma are often differentiated with difficulty, and indeed they are frequently concomitant diseases. However, in pure hypertrophic emphysema the expiratory prolongation and the attendant physical signs are constant and are not paroxysmal in their manifestation. Moreover, in emphysema the vital capacity of the lungs is permanently diminished, whereas in bronchial asthma in the intervals between the paroxysms this capacity closely approximates the normal, to become abruptly impaired during the asthmatic attack. Moreover, the microscopic examination of the sputum in bronchial asthma reveals the presence of characteristic elements. As to chronic bronchitis and bronchial asthma, the former may be assumed to be present when in the intervals between the paroxysms physical signs of moist bronchial flammation are persistently discovered upon successive exami-
is.

TRACHEOBRONCHIAL STENOSIS

Clinical Pathology.—Diminution of the lumen of the tracheobronchial system arises from intrabronchial and from extrabronchial causes. Intrabronchial stenosis occurs with tracheal and bronchial diphtheria, as the result of the development and growth of polypoid tumors of the mucous membrane, as a consequence of turgescence of the mucosa following the inhalation of irritating gases, in cicatricial formations following syphilitic ulceration, and in the presence of foreign bodies. The trachea may be compressed above the bifurcation by an enlarged thyroid gland or a mediastinal tumor, as also in the course of caries of the vertebral column and by extension of esophageal disease. Below the bifurcation the tracheobronchial tree is liable to compression from aortic aneurysm, pericardial effusion, or an excessively hypertrophied heart, pulmonary tumor, or enlarged bronchial glands. Aneurysm of the ascending aorta usually compresses the right bronchus, whereas aneurysm of the arch or of the descending aorta causes compression of the left bronchus.

The ultimate effect of the stenosis upon the lungs varies with the degree and with the site of the obstruction. Stenosis above the tracheal bifurcation results in deficient, but prolonged expansion of the lungs. Total and permanent obstruction of a principal bronchus results in ultimate collapse of the lung which it supplies. Complete stenosis of a bronchial tube of the second or third order results in atelectasis of a considerable area of a lung; while stenosis of several of the small bronchi exerts little influence upon the lung as a whole, as the area of atelectasis is compensated by vicarious expansion of the adjacent pulmonary lobules.

Upon complete stenosis of any portion of the tracheobronchial system, the air in the infundibula supplied by the affected tube is gradually absorbed and the pulmonary lobules collapse, constituting the so-called *obturation atelectasis*. The lobules immediately adjacent to an area of atelectasis are compensatorily emphysematous, and by their vicarious expansion and increased aerial content mask the physical signs which would naturally be produced by the area of atelectasis.

Physical Signs.—The physical signs of tracheobronchial stenosis vary with the site and the degree of the obstruction. In tracheal obstruction the signs are bilateral in their distribution, affecting both lungs to an equal degree; whereas in stenosis of a principal bronchus the physical manifestations of the condition are unilateral, but affect an entire lung. In stenosis of a small bronchus physical signs are slight in degree or are entirely absent.

Inspection.—In tracheal stenosis the clinical picture is that of inspiratory dyspnea with inspiratory retraction of the lower intercostal spaces and epigastrium, and with bilateral diminution in the thoracic expansion. In extreme grades of tracheal stenosis there is cyanosis of the mucous membranes and extremities. In stenosis of a principal bronchus, expansion is limited over the side of the affected lung. Tracheobronchoscopy is often of service in the detection of stenosis, revealing the site and the degree of the obstruction.

Palpation.—Vocal fremitus is diminished or absent over the distribution of the obstructed tube, the alteration in intensity being bilateral in tracheal stenosis, and unilateral in bronchial obstruction.

Percussion.—In stenosis of a principal bronchus the percussion note is dull over the area of distribution of the bronchus, or over the entire lung. In minor degrees of stenosis the note is scarcely changed, and indeed it may be hyperresonant as a result of the compensatory emphysema of the infundibula adjacent to the area of atelectasis.

Auscultation.—Upon auscultation over the area of an occluded bronchus of considerable magnitude the respiratory murmur is feeble or is entirely abolished, while over the opposite lung it is exaggerated or puerile. Coarse, sonorous râles are occasionally to be elicited over the site of the obstruction. Vocal resonance is abolished over the affected lung in the presence of obstruction of a principal bronchus; but it is little affected in minor degrees of stenosis.

Diagnosis.—The presence of inspiratory dyspnea, sibilant and sonorous râles, confined to a circumscribed area, unilateral deficiency of expansion and dullness over an entire lung, are signs suggestive of obstruction of a principal bronchus. In cases of stenosis above the tracheal bifurcation it is important to differentiate laryngeal from tracheal stenosis. In laryngeal stenosis there is vigorous respiratory movement of the larynx; the head is fixed and thrown somewhat backward; and the respiration is stridulous. The employment of the laryngoscope reveals the constriction or the obstructing body. Tracheal stenosis is attended by less stridor, with limitation of the laryngeal movement during respiration, and frequently the patient is orthopneic. Fluoroscopy reveals the site foreign bodies when these are the underlying cause of the osis.

CHAPTER IX

CIRCULATORY DISTURBANCES OF THE LUNGS

PULMONARY CONGESTION (Congestion of the Lungs)

Clinical Pathology.—Congestion of the lungs occurs in two forms; namely, as *active congestion,* and as *passive congestion.*

Active congestion of the lungs occurs in the early stages of inflammation of these organs, as in the period of engorgement of lobar pneumonia. But active congestion of the lungs frequently occurs in conditions which do not attain the gravity of the first stage of lobar pneumonia; notably after the inhalation of irritating gases, of hot or cold air; and it occurs as a collateral congestion due to disease of an adjacent area of the lung. Active pulmonary congestion while not usually dangerous in itself, has in rare instances terminated fatally. Postmortem the lung in active congestion is enlarged, is deep red, its consistence is increased, yet the lung is crepitant and will float when placed in water.

Passive congestion of the lungs occurs in two forms; namely, as *mechanical congestion,* and as *hypostatic congestion* of the lungs.

Mechanical congestion of the lungs results from an obstacle which is interposed to the free return of blood from the lungs to the heart. The most common cause operating in this manner is mitral regurgitation, while a less frequent cause is a tumor pressing upon the pulmonary veins. Mitral stenosis and aortic insufficiency and stenosis operate similarly to produce mechanical congestion of the lungs.

Hypostatic congestion of the lungs is encountered in adynamic and asthenic states, particularly in elderly subjects who have been long in the recumbent posture during a continued fever or chronic wasting disease. The congestion in this instance is localized to the posterior and inferior portions of the lungs, and is largely dependent upon general asthenia and relaxation of the pulmonary vessels. That it is not entirely due to the supine posture is evinced by the fact that it only occurs in subjects who are weakened by the alveolar spaces contain serous fluid, desquamated alveolar cells containing blood pigment, and a few leucocytes and red blood cells.

Physical Signs.—*Inspection.*—In pulmonary congestion the respiratory movements of the thorax are hurried but are limited in amplitude, the degree of thoracic expansion being below the normal degree. In acute congestion the face exhibits a variable degree of cyanosis; the expression is that of great anxiety; and the alæ nasi move with respiration.

Palpation.—In active congestion palpation reveals a moderate exaggeration of vocal fremitus, which is most readily detected over the bases posteriorly. In passive congestion, on the contrary, there is diminution of the fremitus over this area of the thorax.

Percussion.—In active congestion of the lungs the percussion note is apt to be slightly hyperresonant, as a result of the increased tension of the pulmonary tissues; whereas in passive congestion there is impairment of resonance over the bases posteriorly, owing to the gravitation of the blood to these regions of the pulmonary system.

Auscultation reveals in active congestion bronchovesicular breathing; and, in cases of passive congestion shows in addition the valvular lesion which is responsible for the congestion. In each instance the pulmonic second sound is accentuated as a result of the increased tension in the pulmonary circulation. In passive congestion moist or bubbling râles are not infrequently audible over the bases posteriorly.

Diagnosis.—Dyspnea with anxious facies, in conjunction with a history of violent physical exertion, or exposure to irritant vapors or in the presence of active pulmonary disease, with limited excursion of the thorax is suggestive of acute pulmonary congestion, particularly if the acute symptoms develop abruptly without warning. The presence of dullness, impaired vocal fremitus, and moist or bubbling râles over the bases posteriorly in an asthenic or bed-ridden patient, is suggestive of passive congestion of the lungs; while, if a valvular lesion of the heart is coupled with the respiratory findings, the diagnosis is yet more probable.

PULMONARY EDEMA (Edema of the Lungs)

Clinical Pathology.—Edema of the lungs occurs in an acute and a chronic form and as general and local edema.

Acute pulmonary edema complicates the acute infectious diseases, as during the course of lobar or lobular pneumonia, scarlatina, influenza, acute rheumatic fever, variola, and typhoid fever. Less frequently it has developed during pregnancy and in hysterical patients.

Chronic pulmonary edema occurs as a result of cardiac weakness during the course of myocarditis, valvular disease, chronic interstitial nephritis, or general arteriosclerosis.

General pulmonary edema involves the whole of both lungs, the manifestations of the stasis being more marked in the bases as a result of gravitation of the fluid toward the dependent portions of the lungs. The lung of general pulmonary edema is sodden, and heavy; the alveoli of the bases contain serous fluid, while the interalveolar walls are thickened and edematous. The lung pits upon pressure, and upon section serous fluid exudes from the surface of the section. The alveoli contain numerous desquamated alveolar epithelial cells which contain blood pigment, the so-called "heart-failure cells."

Local pulmonary edema occurs as a collateral edema about foci of active pulmonary inflammation in the course of pneumonia, phthisis, and pulmonary infarction. In these cases the signs of the primary affection in the main obscure those of the local edema.

General pulmonary edema is attended by the expectoration of abundant, clear, thin, frothy sputum, which is raised in large quantities.

Physical Signs.—*Inspection.*—The subject of pulmonary edema is suddenly siezed with dyspnea, which rapidly progresses to orthopnea, with anxious facies, and frequently with cyanosis of the lips and buccal mucosa. Thoracic expansion is diminished in extent, but is increased in frequency. There is constant, harassing cough, the cough of the wet lung, which is attended by the raising of abundant, frothy expectoration.

Palpation.—Vocal fremitus is diminished in intensity over the bases of the lungs, while rhonchal fremitus, the tactile equivalent of the numerous moist râles which are present, is well brought to the fore. The skin of the extremities is moist and cold. The pulse is rapid, with diminution in the volume and force of the waves.

Percussion.—The resonance of the percussion note is impaired over the bases posteriorly, whereas over the anterior surface of the chest in the infraclavicular and mammary regions skodaic resonance is not infrequently encountered.

Auscultation.—The respiratory murmur over the infraclavicular and mammary regions is commonly bronchovesicular, while over the bases vesicular respiration is masked by numerous moist and bubbling râles. The heart sounds are increased in frequency, and exhibit a diminution in their intensity, save that the pulmonic second sound is frequently moderately accentuated. In extreme cases of general pulmonary edema, associated with cardiac dilatation, embryocardia is frequently present late in the course of the disease.

Diagnosis.—The diagnosis of pulmonary edema rests upon the occurrence of dyspnea, frequently amounting to orthopnea, accompanied by impairment of resonance and moist râles over the pulmonary bases, in a subject with an obstacle to the return of blood to the left heart or in an asthenic state from an acute infection or the subject of chronic nephritis or arteriosclerosis.

Pulmonary edema is differentiated from bronchial asthma by the fact that in the former the dyspnea involves both phases of the respiratory cycle, and by the essential characteristics of the expectoration in the two affections.

PULMONARY INFARCTION

Clinical Pathology.—Infarction of the lung occurs as a result of occlusion of one or more terminal branches of the pulmonary arteries. The condition develops most frequently in connection with valvular heart disease, particularly with acute or chronic endocarditis. Pulmonary infarction is almost invariably of the hemorrhagic type.

The areas of infarction are situated most frequently in the lower lobes, and involve the right lung more frequently than the left. Usually of limited extent, a pulmonary infarct may become very extensive, involving the greater part of a lobe of the lung. The infarcts are commonly situated at the periphery of the lung; they are wedge-shaped; and the base of the wedge is directed toward the free surface of the lung. When recent, the areas of infarction are dark red upon section, resembling a blood clot. Later, as organization progresses and the hemoglobin is partially removed by phagocytes, they assume a denser consistence and a yellowish color. Eventually, in the absence of pyogenic organisms, the infarct undergoes organization, leaving a puckered scar at the site of infarction. The pleura overlying an area of infarction exhibits signs of local inflammation.

Microscopically the alveoli of the lung in the area of infarction are filled with erythrocytes in various stages of disintegration.

The vessels of the alveolar walls are likewise filled with red blood cells and in places are apt to exhibit thrombus formation.

The ultimate termination of a pulmonary infarct depends upon the character of the causative embolus. If the embolus which occluded the terminal artery is noninfectious, the infarct gradually undergoes organization, and eventually is converted into a mass of cicatricial tissue at the site of infarction. If, on the contrary, the embolus is of septic origin, the area of infarction is frequently the point of incidence of a pulmonary abscess or of pulmonary gangrene. It is probable that in the case of large areas of infarction, involving the major portion of a lobe of the lung, partial resolution analogous to that which follows the consolidation of lobar pneumonia ensues.

Infarction of the lung is not infrequently attended by the expectoration of viscid, mucoid sputum containing numerous erythrocytes, and occasionally by frank hemoptysis.

Physical Signs.—The signs which are referable to pulmonary infarction are variable, depending as they do upon the number, the size, and the distribution of the infarcts. In the case of small infarcts and of infarction of central portions of the lung, physical signs may be entirely lacking; or the only signs elicited may be referable to the associated pleural inflammation, and may be in no way distinctive of infarction.

Inspection.—In cases of extensive pulmonary infarction the respiratory excursion of the thorax is limited upon the side of the disease. In addition, the subject is apt to exhibit moderate or severe dyspnea, with anxious facies and occasionally hemoptysis.

Palpation.—Palpation may show increased vocal fremitus if the area of infarction is of considerable extent and is situated near the periphery of the lung; whereas, if the infarct is centrally placed, near the root of the lung, no alteration of vocal fremitus is demonstrable. When a peripheral infarct overlies a principal bronchus, vocal fremitus is markedly exaggerated.

Percussion.—Over large infarcts dullness is elicited upon percussion; whereas in the case of a large infarct which is superimposed upon a principal bronchus, the tympany of the bronchus is engrafted upon the dullness of the percussion note. Gerhardt calls attention to the frequency of pulmonary infarction upon the right side in the area of the lung lying between the vertebral column, the diaphragm, and the angle of the scapula, whence the importance in this disease of signs of consolidation over this area.

Auscultation.—In suitably situated infarcts with reference to a main bronchus, loud, bronchial or tubular breath sounds are elicited upon auscultation, as well as râles which are transmitted from the bronchial tube. In cases of multiple infarcts, or of deeply seated infarction, the breath sounds are at the most bronchovesicular, and in many instances they are frankly vesicular.

Pleural friction is frequently to be elicited over areas of superficial infarction; and indeed in the case of infarction of limited extent, involving the peripheral portion of the lung, this is occasionally the only sign of the disease.

Diagnosis.—The physical signs of infarction of the lung are essentially those of pulmonary consolidation, and the diagnosis of infarction is made with difficulty. When signs of consolidation are elicited in a patient suffering with valvular heart disease, from which pulmonary embolism might arise, infarction of the lung becomes a possibility; and, when the physical signs of consolidation are attended by hemoptysis, pulmonary infarction may be assumed to be present. As the area of infarction is frequently situated in the lower lobe of the right lung posteriorly, the physical signs of consolidation simulate closely those of lobar pneumonia.

An infarct of moderate size occupying a central portion of the lung produces no physical signs by which a diagnosis of infarction may be made. A limited, superficial infarct, on the contrary, is apt to produce a local friction rub, which is apt to be mistaken for a simple pleurisy. Septic infarction, if extensive, is attended by signs of septic intoxication, with the supervention of signs of pulmonary abscess or of pulmonary gangrene.

CHAPTER X

DISEASES OF THE LUNGS

LOBAR PNEUMONIA (CROUPOUS, OR FIBRINOUS PNEUMONIA)

Clinical Pathology.—Lobar, croupous, or fibrinous pneumonia is an inflammation of the lung, which is attended by a variable degree of constitutional reaction and toxemia.

The causative microorganism of lobar pneumonia is the pneumococcus or Diplococcus pneumoniæ, first discovered in the sputum of pneumonia patients by Sternberg and Pasteur in 1880, and recognized as the cause of the disease by Fraenkel in 1884. The organism may be found in pure culture in the sputum of pneumonia patients or associated with the streptococcus, staphylococcus, or Friedländer's bacillus. Lobar pneumonia is very prevalent at the extremes of life, affecting especially young infants and elderly persons; but no age is exempt from the disease. The majority of cases develop during the late winter and the early spring months the portion of the lung which is the site of the disease is solid, firm, and devoid of air. The lung is slightly enlarged, and frequently the surface presents indentations corresponding to the ribs with which it is in contact. Upon section, the surface of section is dry, reddish or brown in color, and very friable. Upon scraping the cut surface with the knife, small fibrinous plugs are apt to come away from the cut surface of the lung. The lung is not crepitant in the area of the disease; and a section of the lung will sink when placed in water. Microscopically the alveoli are filled with a dense, fibrinous exudate, which contain erythrocytes, leucocytes, bacteria, and desquamated epithelial cells, embedded and entangled in a matrix of fibrin, the erythrocytes predominating during this stage of the disease.

In the stage of gray hepatization the pulmonary tissue loses its reddish color upon section, the surface of section presenting a gray or grayish-white appearance. Section shows a moister surface than in the preceding stage, and but few fibrinous plugs come away upon scraping the surface with the knife. Microscopically, polymorphonuclear leucocytes predominate the picture, though a variable number of erythrocytes, as well as desquamated epithelial cells and bacteria are present in the fibrinous mass. However, notwithstanding the partial clearing of the alveoli, the lung is still noncrepitant, and a section of the diseased area will sink when placed in water.

During the stage of resolution, if it occurs, the fluid is drained from the lung by the lymphatics; the debris is removed by phagocytes, and is also expectorated with the sputum; and the lung gradually resumes its normal structure. The area of disease once more becomes crepitant, and a section floats when placed in water.

The expectoration during the active stage of lobar pneumonia is thick and viscid, and of a brownish color, the so-called "prune juice sputum." Frequently the viscosity is so great that the sputum will not separate from its container when the latter is inverted.

During the inflammation of the lung in lobar pneumonia the pleura is practically always involved over the area of consolidation, becoming coated with a variable quantity of fibrinous exudate, and not infrequently pouring out a moderate amount of serous fluid into the pleural cavity. However, in central pneumonia, in which the consolidation is deeply situated near the root of the lung, the pleura escapes. It is, however, questionable whether lobar pneumonia ever remains central, or whether every lobar pneumonia which has its point of inception near the root of the lung eventually does not progress and involve the periphery of the lung.

Lobar pneumonia, as the name implies, usually involves the major portion of a lobe or an entire lobe of the lung. The disease is in the majority of instances unilateral. In relative frequency the different portions of the lungs are involved in the following order: lower right lobe, lower left lobe, upper right lobe, an entire lung, or rarely both lungs.

According to the distribution of the disease several clinical types of lobar pneumonia are recognized:

Apical pneumonia affects only the apex of the lung.

Migratory pneumonia successively involves lobe after lobe of the lung in regular progression.

Double pneumonia involves both lungs simultaneously.

Massive pneumonia is a form of the disease in which, in addition to the alveoli, the bronchial tubes of an entire lobe or lung are plugged with fibrinous exudate.

Central pneumonia is a form in which the disease is situated deeply at the root of the lung, and does not at once involve the peripheral portions.

While lobar pneumonia, under favorable circumstances, terminates in resolution with removal of the debris of the consolidation, the disease may terminate by sclerosis, resulting in chronic interstitial pneumonia, while in other instances the morbid process goes on to the formation of pulmonary abscess or gangrene.

Physical Signs.—*Inspection.*—The decubitus of the patient is often suggestive in lobar pneumonia. He may be found lying upon the diseased side or may be found sitting up in bed with the spine curved toward the side of the disease. Herpes labialis is a very common finding in lobar pneumonia; and a red spot or flush upon the cheek of the side of the disease is not an infrequent sign of the disease. The respirations are short, and are frequently accompanied by an expiratory grunt.

In a case of unilateral pneumonia, and the disease in the vast majority of cases is unilateral, inspection reveals restriction of the excursion of the thorax upon the side of the disease, with exaggerated excursion of the sound side. The diseased side does not expand to its normal physiologic capacity for two reasons: the air space in the lung is actually decreased by consolidation; and, moreover, the pleurisy accompanying the pneumonic process causes the patient to inhibit the respiratory movements as much as possible. The sound lung, on the contrary, expands vicariously in the effort to compensate as far as may be for the deficiency of expansion of the diseased lung. Litten's diaphragmatic shadow is abolished upon the affected side of the thorax.

In cases in which the left lung is involved anteriorly, the cardiac impulse is frequently unduly prominent and extensive, as the portion of this lung which overlaps the heart is enlarged and more or less firm and is pushed forward before the heart with each impact of that organ during ventricular systole.

Palpation.—Upon palpation of the thorax over the area of consolidation in the stage of red hepatization vocal fremitus is markedly exaggerated. This increase of fremitus over the base posteriorly, where it is usually encountered in lobar pneumonia, is very striking since under normal conditions of the lung, the fremitus is very faint in this region. The fremitus presents little alteration during the period of engorgement; it reaches its maximum intensity during the stage of red hepatization; and it gradually resumes the normal intensity with the supervention of resolution.

There are two conditions under which vocal fremitus is entirely abolished over an area of consolidation in lobar pneumonia. If the main bronchus leading into this area becomes plugged with fibrinous exudate, as is frequently the case in massive pneumonia, the vocal vibrations are not appreciable to the palpating hand. Again, if there be extensive involvement of the pleura with considerable effusion, the fluid effectually masks the otherwise palpable vibrations during phonation.

During the stage of engorgement and the early days of the stage of red hepatization the pulse is full and bounding, the heart acting powerfully as a result of the raised blood pressure in the pulmonary circuit. In the later stages of the disease, in asthenic subjects, and when the constitutional toxemia of the disease is excessive, the heart is prone to undergo more or less severe parenchymatous myocarditis, the pulse then becoming rapid, running, and feeble.

Percussion.—During the period of engorgement, during the first twenty-four hours of the disease, percussion yields frequently hyperresonance of the Skodaic type. Percussion during the stage of red hepatization reveals dullness or flatness over the consolidated lobe, while percussion immediately above the consolidation, on the contrary, yields Skodaic resonance, due to relaxation of the tissues which are compressed by the consolidation.

In a case of central pneumonia, percussion reveals only vesicular resonance; or, at most, only slight impairment of resonance, because the lung immediately beneath the chest wall is not consolidated. It is here that deep percussion occasionally reveals the presence of the deep consolidation. During the latter portion of the stage of gray hepatization and during the stage of resolution, the lung exhibits a gradual return to the normal vesicular resonance over the area of disease.

Auscultation.—During the period of engorgement the breath sounds are quiet and partially suppressed, while at the completion of full inspiration there occurs a very valuable physical sign; namely, a fine crepitant râle, the crepitus indux. This râle is produced by the separation of the walls of the infundibula, which have become adherent by tenacious secretion, and it presents to the ear of the examiner a shower of fine crackling sounds.

When the consolidation is fully established, in the stage of red hepatization, the crepitus indux is replaced by distinct bronchial or tubular breathing. During the latter portion of the stage of gray hepatization and during the period of resolution, when the consolidation becomes macerated and partially dissolved, the bronchial breath sounds are replaced by bronchovesicular breathing and subcrepitant râles, constituting the crepitus redux of this disease.

The pulmonic second sound is accentuated as a result of the increased load which is thrown upon the right heart as the result of the obstacle which is offered to the passage of the blood through the pulmonary circulation. In prolonged cases of lobar pneumonia there is frequently a reduplication of the second sound of the heart due to asynchronous closure of the aortic and pulmonic valves, caused by the unequal tension in the general and the pulmonary circulations.

Diagnosis.—In a case of frank lobar pneumonia in an adult, with abrupt onset with pain in the side, initial chill, and rapidly rising fever, coupled with the development of rusty tenacious sputum, the diagnosis of lobar pneumonia is not difficult. But in young children, in the aged, in alcoholic subjects, and in terminal or secondary pneumonias which are engrafted upon other conditions as carcinoma, nephritis, or diabetes, the diagnosis is often reached with considerable difficulty.

In a case of frank uncomplicated lobar pneumonia the physical signs are definite and distinctive; lessened or deficient expansion of the diseased side of the thorax, exaggeration of vocal fremitus, dullness or flatness upon percussion, and bronchial breathing, with the crepitant râle on auscultation. But it should be borne in mind that in massive pneumonia vocal fremitus is apt to be diminished or abolished over the diseased area owing to plugging of the principal bronchus supplying the part. Moreover, in ordinary pneumonia, in the routine case, the dullness is preceded and followed by a vesiculotympanitic note, occurring prior to and following complete hepatization of the pulmonary tissues in the area of the disease. So also the bronchial breath sounds are absent or atypical in the presence of incomplete hepatization of the lung, in partial plugging of a bronchus, and in the presence of a complicating pleurisy with effusion. Additional data for diagnosis are the facts that the fever commonly terminates by crisis at the seventh or ninth day; that the onset is very abrupt; that the pulse-respiration ratio is markedly altered; that herpes labialis is particularly frequent in this disease; and that there is commonly a hectic flush upon the cheek upon the side of the disease.

The localization of the disease in the lung is prone to influence the physical signs and to occasion difficulties in diagnosis. In massive pneumonia the ordinary physical signs are apt to be lacking over a large area of the lung, giving physical signs simulating pleurisy with effusion. In this class of cases it is occasionally possible to dislodge the plugging exudate by coughing.

Central pneumonia fails to give the typical signs of frank lobar pneumonia. Here the lesion, starting deeply within the lung, overlaid as it is by normal pulmonary tissues, yields a bronchovesicular respiratory murmur and only moderate impairment of pulmonary resonance, if indeed there is any. Deep percussion in these cases may serve to elicit dullness. However, as a rule, these cases can only be said to be central in their incipiency, as they usually eventuate in a peripheral lobar pneumonia involving one or more lobes of the lung.

Lobar pneumonia is not infrequently a source of pain in the right lower portion of the abdomen, thus simulating acute appendicitis. In other instances the disease is attended by constipation, abdominal pain and meteorism, simulating intestinal obstruction. In drunkards the cerebral symptoms of pneumonia predominate; while in children cerebral symptoms are prominent and the rusty sputum is frequently absent throughout the course of the disease.

Differential Diagnosis.—Lobar pneumonia must be differentiated from acute pneumonic phthisis, bronchopneumonia, pulmonary infarction, pulmonary edema, pulmonary congestion from cardiac defects, and from pleurisy with effusion.

Acute Pneumonic Phthisis.—In the early stages of the disease it is often impossible to differentiate between this disease and lobar pneumonia. This form of pulmonary tuberculosis begins abruptly with chill, pain in the side, and cough which is attended by sputum which is at first mucoid, and later rusty. The physical signs are those of consolidation of one or more lobes, or possibly of an entire lung. The chill is followed by a rapid rise of fever, and the picture is for a time that of frank lobar pneumonia. But at the seventh or ninth day no crisis occurs; on the contrary, the fever persists, and is attended by night sweats, while elastic fibers and tubercle bacilli appear in the sputum. Evidences of softening soon become manifest, with moist or gurgling râles and signs of cavitation.

Bronchopneumonia.—Bronchopneumonia is usually gradual in onset and is usually secondary to other infectious fevers, as measles, influenza, scarlatina, or typhoid fever; or it occurs as the result of the aspiration of material from disease of the upper respiratory passages, or from the decomposition of the contents of bronchiectatic dilatations.

The distribution of the disease is of assistance in differentiation, bronchopneumonia being bilateral, whereas lobar pneumonia, as a rule, affects one lobe or one lung. Moreover, the age at which the two diseases are most prevalent differs. Bronchopneumonia is most frequently encountered in young subjects, under three years of age frequently, whereas lobar pneumonia is more common after the third year and in elderly subjects.

The physical signs of bronchopneumonia are diffuse, the patches of consolidation being scattered widely over both lungs, so that the percussion note is never frankly dull or flat, but there is bilateral impairment of pulmonary resonance of moderate degree. Similarly, bronchopneumonia is not attended by frank bronchial breathing, but by bronchovesicular breath sounds. Crisis does not occur in bronchopneumonia, the fever resolving by lysis.

Pulmonary Infarction.—Infarction of the lung is abrupt in onset, with dyspnea and cough, and the expectoration is not viscid, but is fluid and often tinged with blood or the disease is attended by frank hemoptysis. In simple infarction the febrile movement does not equal that of lobar pneumonia, but in septic infarction a local pneumonia frequently develops at the site of the infarct, and results in pulmonary abscess with its hectic temperature and colliquative sweats. There are often signs of an associated valvular heart lesion, as the disease often occurs in persons with chronic endocarditis.

Unless they are very extensive, pulmonary infarcts produce very few definite physical signs, though extensive infarction of the lower lobe of the lung produces bronchovesicular or bronchial breathing when properly situated with reference to a large bronchus.

Pulmonary edema develops commonly in patients with valvular heart lesions or nephritis, producing extreme dyspnea or orthopnea, with numerous moist or bubbling râles over the bases posteriorly. The respiratory sounds are diminished in intensity rather than purely bronchial. The sputum is abundant and characteristic; and the disease in its manifestations is bilateral, affecting the bases of both lungs, and it is not attended by fever.

Pulmonary Congestion.—The variety of pulmonary congestion which most closely simulates lobar pneumonia is the acute active congestion which is described by the French authors as Woillez's disease, a form of active congestion of sudden onset, which really constitutes a larval type of pneumonia. Hypostatic congestion is bilateral in its manifestations, with moist râles at the bases of the lungs; but there is no fever, and the sputum is not viscid or rusty.

Serofibrinous pleurisy with effusion may be confused with lobar pneumonia; but there are sufficient diagnostic points to differentiate the two diseases with comparative ease.

In pleurisy with effusion the onset is gradual, with chilly sensations rather than abrupt with a distinct chill as in the case of lobar pneumonia. Pleurisy in the early stage is almost always accompanied by a pleural friction sound, which disappears as the effusion accumulates in the pleural cavity. Pleuritic fever resolves by lysis, while the fever of lobar pneumonia terminates by crisis. In pleurisy with effusion there is often a tuberculous history obtainable in connection with the pleurisy. In pleurisy with effusion vocal fremitus is abolished, instead of exaggerated as is the case in lobar pneumonia. There is flatness upon percussion, whereas in pneumonia the note is dull or flat, but is preceded and followed by a hyperresonant note during the stages of incomplete hepatization. Instead of the crepitant and subcrepitant râles of pneumonia, in pleurisy with effusion there is total abolition of respiratory sounds as a rule over the effusion. Aspiration of the thorax reveals fluid in the case of pleurisy with effusion.

Herpes labialis is rare in pleurisy, and very common in lobar pneumonia. Egophony may be elicited below the scapular angle with fair constancy in pleurisy with effusion; and the cardiac apex beat is displaced to the side opposite to the effusion. The intercostal spaces are more apt to be obliterated or to bulge in pleurisy with effusion than in pneumonia. Visceral displacement is always more pronounced in pleurisy than is the case with lobar pneumonia.

BRONCHOPNEUMONIA (LOBULAR OR CATARRHAL PNEUMONIA)

Clinical Pathology.—Bronchopneumonia, catarrhal, or lobular pneumonia is an acute inflammation of the terminal bronchioles, spreading secondarily to the adjacent alveoli, which become filled with inflammatory exudate from the terminal bronchioles.

Bronchopneumonia is almost invariably a secondary disease, following many of the infectious fevers, as pertussis, measles, influenza, diphtheria, scarlatina, variola, or typhoid fever, diseases which during their course have been associated with a greater or less degree of acute bronchitis. In these instances the development of bronchopneumonia merely represents a downward extension of the acute bronchitis to the finer bronchioles, where it constitutes a capillary bronchitis.

Bronchopneumonia also follows the aspiration into the bronchi of particles of food, or of secretions, or of blood from the upper respiratory passages (aspiration or deglutition pneumonia). In apoplexy, and during comatous states from other causes, particles are prone to be aspirated into the bronchi and to set up a bronchopneumonia. Aspiration pneumonia occurring in the newly born is due to the aspiration of secretions from the birth canal during labor.

Bronchopneumonia is very common in infancy, attacking many children under one year of age. In this class of patients the presence of rickets or severe diarrhea predisposes to the development of the disease.

A primary bronchopneumonia occasionally develops in adults and children who are below par, in which the onset of the disease is abrupt, simulating in this respect lobar pneumonia.

The organisms which are most frequently responsible for bronchopneumonia are Friedländer's bacillus and the pneumococcus of Fraenkel. These organisms may occur alone or in association with the streptococcus, influenza bacillus, colon bacillus, or staphylococcus

In bronchopneumonia the lung presents upon its surface scattered patches of consolidation, which are separated by areas of compensatory emphysema. The consolidated patches are red or grayish, and slightly elevated above the surrounding surface of the lung. The disease is bilateral, affecting both lungs, which remain crepitant despite the multiple areas of consolidation, and the lung will float when it is placed in water.

When the lung is sectioned, the pneumonic patches of consolidation are observed to be situated in and confined to the peripheral portions of the lung, just subjacent to the pleura.

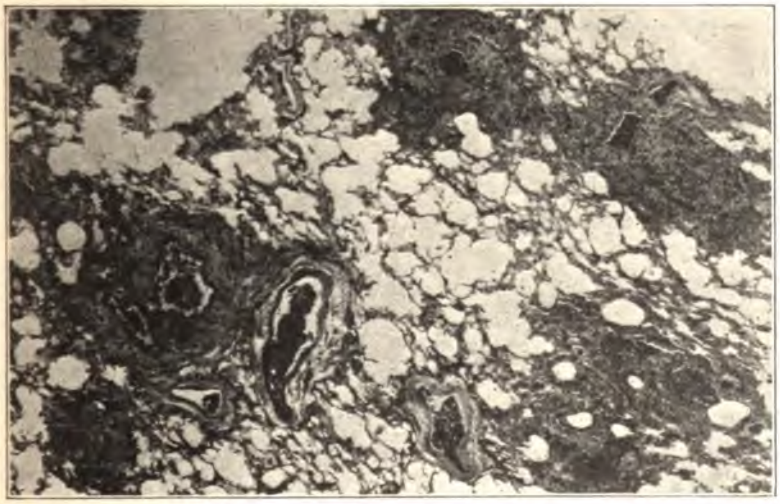


Fig. 106.—Consolidation of bronchopneumonia. (From Delafield and Prudden.)

The terminal bronchioles are filled with mucopurulent exudate; while the peribronchial tissues are consolidated in the immediate vicinity of the bronchus, and exhibit splenization for some distance beyond the area of consolidation. Microscopically the bronchial walls present swelling and desquamation of the lining cells, while the bronchial lumen is filled with mucopurulent exudate, composed mainly of desquamated bronchiolar cells and with very few erythrocytes and leucocytes. The bronchial walls and interalveolar septa in the consolidated areas show infiltration with leucocytes.

The distribution of the disease varies. As the name implies, the disease has a tendency to involve lobules rather than lobes of the lung. As a rule, this principle holds true in the distribution of the lesions, which usually assume one of two types. Thus, in the disseminated form of bronchopneumonia lobules are consolidated throughout the two lungs, the areas of consolidation being separated by areas of crepitant lung. In the pseudolobar form, however, a number of lobules in one lobe are consolidated, perhaps affecting the greater portion of a lobe of the lung.

Bronchopneumonia may terminate in resolution, in abscess, in fibrosis, or in gangrene. In the cases which terminate by resolution the cellular exudate in the bronchioles and alveoli becomes liquefied, largely as a result of fatty degeneration, and is borne away by the lymphatics or is expectorated.

The pleura is frequently involved in bronchopneumonia, but not to the same degree as in lobar pneumonia; but when pleurisy develops with bronchopneumonia it is very apt to be of the purulent type.

The expectoration in bronchopneumonia is tenacious and glairy, containing no characteristic elements.

Physical Signs.—*Inspection.*—Bronchopneumonia is constantly attended by signs of obstruction of the terminal bronchioles, as evinced by inspiratory dyspnea, with participation of the accessory muscles of respiration and inspiratory retraction of the lower intercostal spaces and epigastrium. In infancy and childhood there is frequently cyanosis of the mucous membranes and extremities, with suffocative attacks; and in infancy the disease is occasionally ushered in with convulsions. In the fully established case of bronchopneumonia the thoracic expansion is bilaterally restricted; and, moreover, in instances of extensive consolidation of the bases the upper portion of the thorax exhibits a greater degree of expansion than does the lower portion. Atelectasis of the anterior border of the left lung, which overlies the heart, results in a cardiac impulse of increased extent, whereas compensatory emphysema of the left pulmonary borders causes a corresponding diminution in the area of cardiac pulsation in other cases.

Palpation.—Palpation reveals increase of vocal fremitus when there is a patch of consolidation of sufficient size and favorably situated with reference to a bronchus. However, the intervening emphysematous portions of the lung tend to mask the fremitus, which may be actually diminished. Similarly, occlusion of a bronchus by exudate may abolish the fremitus. In the pseudolobar form of the disease there is invariably increase of vocal fremitus, whereas in the disseminated form such an increase is not to be expected.

During the early stage of the disease the pulse rate is accelerated, with full volume and normal rhythm. During the late stage of the disease, in unfavorable cases, the rhythm of the pulse is disturbed, the pulse becoming rapid, readily compressible, and often dicrotic.

Percussion.—In bronchopneumonia during the first twenty-four hours of the disease, percussion of the thorax elicits only normal pulmonary resonance; but in a suspected case the bases posteriorly should be percussed daily; and in most instances at the expiration of forty-eight hours one or more areas of dullness will have become demonstrable in these regions. However, the thorax of the young child, in whom the disease is frequently encountered, is normally slightly hyperresonant; and, moreover, the areas of compensatory emphysema, which surround and separate the patches of consolidation, tend to mask any dullness which would be produced by the presence of the consolidated areas, and to impart a vesiculotympanitic quality to the percussion note. In the disseminated form of bronchopneumonia frank dullness is not to be expected, as the hyperresonance of the associated compensatory emphysema effectually masks the dullness of the consolidation. In the pseudolobar form, on the contrary, dullness may always be elicited by careful daily percussion of the bases posteriorly. In demonstrating the dullness of superficial patches of consolidation it is essential to employ very light superficial percussion; while in eliciting the dullness of deeply seated areas of solidification more forcible, deep percussion should be employed.

Percussion of the anterior portion of the thorax in the infraclavicular and mammary regions yields Skodaic resonance. The presence of bilateral dullness over the bases posteriorly with Skodaic resonance anteriorly is very suggestive of the consolidation of bronchopneumonia.

Auscultation.—Auscultation reveals upon consecutive examinations the downward extension of the original bronchitis. In addition to the râles arising in the bronchial tubes incident to the bronchitis, the crepitant râle is audible upon the completion of inspiration over the bases posteriorly, indicating the participation of the alveoli in the inflammatory process. At times the râles are obscured by plugging of the bronchioles by inflammatory exudate; but coughing usually serves to bring them again to the fore.

As the patches of consolidation form, the respiratory sounds assume a bronchovesicular character; but they never become purely bronchial as is the case in lobar pneumonia. The pulmonic second sound is accentuated during the height of the disease; and if the right heart fails, edema of the lungs supervenes, with the characteristic signs of that condition.

In the presence of considerable consolidation of the bases, bronchophony is occasionally to be elicited just above the level of the consolidation. Over the anterior surface of the thorax, in the region of compensatory emphysema, the expiratory murmur is slightly prolonged, and is accompanied by piping, sibilant râles.

Diagnosis.—While the physical signs of bronchopneumonia are bilateral in distribution, they are seldom present to the same degree upon both sides of the thorax, one lung usually being involved to a greater extent than is the other. Whenever physical signs of consolidation are elicited over one lung, a careful search for similar signs should be made over the opposite lung.

Very frequently a diagnosis of bronchopneumonia cannot be made upon the physical signs alone, as these are often misleading; and as the mode of onset of the disease is not infrequently atypical. The physical findings should be considered in conjunction with the fever, cough, dyspnea, and pain in the thorax, arising during the course of an acute infectious disease, or engrafted upon a previously existing acute bronchitis. Moreover, occurring as the disease does during the course of, or convalescence from, another acute disease, the clinical picture is often modified for a time at least by the characters of the primary affection. But a history of fever, with dyspnea, cough, and pain in the chest, arising during the course of an acute infection or engrafted upon a previous bronchitis is suggestive of a complicating bronchopneumonia.

Differential Diagnosis.—From *acute bronchitis* the disease is differentiated by the absence of areas of hyperresonance and impaired resonance in the former, and by the finer quality of the râles in bronchopneumonia. Moreover, in acute bronchitis there is little or no fever, and the disease is altogether milder in its manifestations.

From *lobar pneumonia,* bronchopneumonia presents many points of differentiation. Bronchopneumonia is usually secondary to another disease and is of insidious onset; whereas lobar pneumonia is usually a primary disease, with abrupt onset and stormy course. Moreover, bronchopneumonia is a bilateral pulmonary disease, producing multiple areas of impaired resonance over both lungs, rather than a single area of dullness or flatness over one or more lobes of a lung. Also, the fever of bronchopneumonia ter
gradually by lysis, whereas that of lobar pneumonia te

crisis between the seventh and the ninth days in the majority o cases. As a rule, bronchopneumonia attacks subjects of impaire vigor who are below par physically, whereas lobar pneumoni frequently attacks robust persons in the prime of health.

In the rarely encountered primary form of bronchopneumonia and in those cases in which the disease assumes the pseudoloba form, in which the pneumonic patches fuse and involve the greate portion of a lobe of the lung, the differentiation from lobar pneumonia is attended with considerable difficulty. In this class of case the physical signs are very similar if not identical; but in bronchopneumonia there is almost invariably apparent, even if to a sligh degree, some involvement of the opposite lung. Moreover, in loba pneumonia the sputum is fairly characteristic, differing markedl from the viscid, glairy expectoration of bronchopneumonia.

From *acute bronchopneumonic phthisis*, bronchopneumonia cannot be differentiated in the incipient stage. Involvement of th apices by the disease is suggestive of phthisis; but in certain case of bronchopneumonic phthisis the apical involvement is not particularly prominent. However, in phthisis the temperature i prone to remain more uniformly high, and to be interrupted earl in the course of the disease by night sweats. There is usually obtainable a history of intimate association with a tuberculous perso or evidence of a tuberculous focus at some point. Moreover, i acute bronchopneumonic phthisis emaciation progresses rapidly the course of the disease being progressively downward, unti eventually tubercle bacilli are demonstrable in the sputum.

Bronchopneumonia in infancy is sometimes with difficulty differentiated from meningitis, as the disease in this class of patients i often accompanied by marked cerebral symptoms, as delirium o convulsions. Time and observation are essential to a differentia diagnosis under these circumstances.

CHRONIC INTERSTITIAL PNEUMONIA (PRODUCTIVE PNEUMONIA; CIRRHOSIS OR FIBROSIS OF THE LUNG)

Clinical Pathology.—Chronic interstitial pneumonia, productive pneumonia, cirrhosis or fibrosis of the lung, is a chronic in ive pulmonary disease, the ultimate result of continue n of various areas of the lung. The predominant featur e is the formation of an excess of fibrous connectiv tissue, which by subsequent contraction decreases the size and the vital capacity of the lung.

Chronic interstitial pneumonia occurs in three forms; namely, the *massive* or *lobar form;* the *insular*, or *bronchopneumonic form;* and the *pleurogenous form* of the disease. To these three principal groups may be added the cases of local or circumscribed pulmonary fibrosis, which result from the inhalation of irritant dusts, or which develop in the evolution of pulmonary tuberculosis and syphilis of the lung.

The *massive or lobar form* of the disease is almost invariably a unilateral affection, involving the lower lobe to a greater degree than the upper lobe of the lung. In the rarer instances of bilateral involvement the induration is much more pronounced in one lung than in its fellow. Massive chronic interstitial pneumonia occurs as a result of incomplete resolution after lobar or after bronchopneumonia, as a terminal change in extensive atelectasis, and occasionally as a result of prolonged compression of the lung by chronic pleural effusion. In this form of the disease the pleura is but slightly involved. The interalveolar septa are greatly thickened by the formation of an excess of fibrous connective tissue, and the alveoli are reduced in size

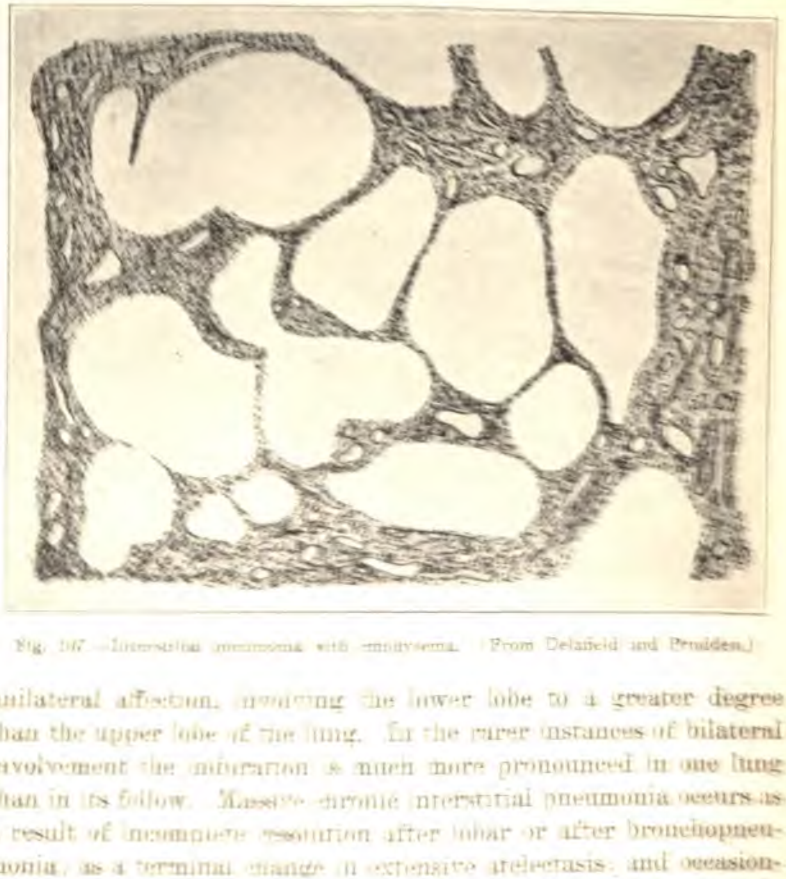


Fig. 107.—Interstitial pneumonia with emphysema. (From Delafield and Prudden.)

In the *insular* or *bronchopneumonic form* of the disease the induration has its inception as a peribronchial fibrosis, involving principally the lower lobe, though the upper lobe is not infrequently involved as well. By traction of the fibrous bands bronchiectatic dilatations of variable dimensions are produced during the evolution of the disease.

Pleurogenous interstitial pneumonia develops as a consequence of pulmonary compression by chronic pleural effusion. The pleura is excessively thickened, and fibrous bands pass from the visceral pleura along the interalveolar septa to the bronchi in the deeper portions of the lung.

The ultimate result of chronic pulmonary fibrosis is very similar in the three types of the disease. The indurated lung is shrunken, hard, and diminished in size, occupying a position in the upper portion of the pleural cavity near the vertebral column. Dense adhesions are apt to form between the visceral and parietal pleuræ, binding the lung to the chest wall. Upon section, bronchiectases of variable dimensions are encountered, in various stages of evolution, ulceration, or abscess formation.

Physical Signs.—*Inspection.*—In chronic interstitial pneumonia the thorax upon the side of the disease is restricted in its expansion, and occasionally it is immobile during the respiratory excursion of the opposite side. Retraction of the side with drooping of the shoulder is noted in advanced cases of the disease. The cardiac impulse is displaced toward the affected side; and when the disease attacks the left lung there is not uncommonly a very wide area of pulsation in the third and fourth interspaces. As a result of the retraction of the diseased side, the nipple and the scapula approach more nearly to the median line than is normal The spinal processes present lateral curvature, with the concavity directed toward the side of the cirrhotic lung.

In cirrhosis of the right lung the cardiac impulse is frequently invisible, as it is not infrequently displaced so far toward the right as to lie behind the sternum. Even in those cases in which the right-sided cirrhosis is not so great as to displace the impulse behind the sternum, the apex-beat may yet be invisible owing to overlapping of the apex of the heart by the compensatorily emphysematous left lung.

Palpation.—In the vast majority of cases of chronic interstitia pneumonia vocal fremitus is exaggerated upon the side of the disease, although in cases of pleurogenous origin which are associated with excessive pleural thickening, it may be diminished

In these cases a pleural friction fremitus can occasionally be detected upon palpation. Vocal fremitus is very greatly increased when a large bronchial dilatation approaches the chest wall, which occurs most frequently in the axillary region.

Percussion.—As a rule, percussion of the thorax over the area of the retracted lung yields a dull or flat note; but percussion of the upper axillary region in many instances yields tympany due to the close proximity to the chest wall of a bronchiectasis. Percussion of the sound lung yields a moderately hyperresonant note, as the result of compensatory emphysema. The superior limit of pulmonary resonance is diminished on the side of the disease as a result of retraction of the apex of the lung. Similarly, in involvement of the right lung the lower border of pulmonary resonance is elevated, the liver pushing the diaphragm upward; whereas in left sided disease the tympany of Traube's semilunar space extends to an abnormally high level.

Auscultation.—Auscultation of the affected side reveals bronchial breathing over the retracted and shrunken lung, which, over dilated bronchi or bronchiectatic cavities, frequently has an amphoric or cavernous quality engrafted upon it. Over the lower portion of the thorax of the affected side, and in cases which are associated with excessive pleural thickening, the respiratory murmur is distant or is entirely abolished. Over the pulmonary apex and in the axillary region, where the bronchial dilatations are apt to approach most nearly to the chest wall, the respiratory murmur is bronchial or amphoric. Over the sound lung the respiration is puerile as the result of compensatory emphysema. In cases of pleurogenous origin a pleural friction sound is frequently audible over the cirrhosed lung.

The pulmonic second sound is accentuated; and late in the course of the disease is apt to become weakened, the tricuspid systolic "safety-valve" murmur then becoming audible, betokening imminent right heart failure.

Diagnosis.—In a subject presenting unilateral retraction of the thorax with drooping of the corresponding shoulder and signs of pulmonary collapse of extensive duration, following pneumonia or chronic serofibrinous pleurisy, chronic interstitial pneumonia is suggested. Early diagnosis of the disease is rendered difficult by the fact that limited areas of fibrosis, whether situated peripherally or centrally, yield few physical signs. A slowly growing neoplasm of the lung or pleura closely simulates the physical signs of chronic interstitial pneumonia.

It is to be borne in mind that the physical signs of fibroid phthisis are very similar to those of chronic interstitial pneumonia. In phthisis, however, the disease is more frequently bilateral, and the persistence of moist râles at an apex speaks for phthisis. The detection of the tubercle bacillus in the sputum is in the end the deciding factor in the differential diagnosis of the two diseases.

TUBERCULOSIS OF THE LUNGS

In the incidence of pulmonary tuberculosis the tubercle bacillus may reach the lung directly through the inhalation of the dried sputum of a tuberculous patient or by droplet infection A break in the continuity of the mucous membrane of the respiratory tract is not essential for infection with the tubercle bacillus; but catarrhal inflammation of the air passages predisposes to infection with the organism.

Aside from direct, aerial infection, pulmonary tuberculosis has followed the ingestion of infected food. In this method of infection it is possible that the bacilli are absorbed through the tonsils, passing primarily to the cervical lymph nodes; but in the majority of instances the organisms reach the intestine whence they pass through the mucous membrane and enter the lymphatics which form the radicles of the thoracic duct, ultimately reaching the blood stream and causing general infection with the tubercle bacillus.

Race, social environment, and the state of health of the individual all influence the incidence of pulmonary infection with the tubercle bacillus. The American Indian and the Negro exhibit a marked susceptibility to pulmonary tuberculosis, though in the latter race it is difficult to separate the racial predisposition to the disease from the effects of social environment. Doubtless the most important single contributing factor is that of the environment in any case, a factor which accounts in many instances for the so-called family predisposition to the disease. Catarrhal inflammation of the respiratory tract incident to pneumonia, pertussis, measles, and influenza predispose to infection with the tubercle bacillus; whereas constitutional diseases, as diabetes mellitus and symptomatic anemia, render the patient also more subject to the disease. Aside from the production of a soil which is favorable for the growth of the organism, these diseases are apt to call into activity a latent or residual tuberculous lesion of the lung.

The primary lesion of pulmonary tuberculosis is a minute, grayish mass of inflammatory tissue, the miliary tubercle. By the growth of the miliary tubercle and by the coalescence of adjacent tubercles more extensive areas of tuberculous infiltration and consolidation arise within the pulmonary tissues. As a result of the histological structure of the tubercle, as the mass extends by peripheral growth, the central portion, representing the original tubercle, becomes ischemic and tends to undergo softening and caseation. The usual termination of an area of caseation is pulmonary excavation, containing caseous material which, by admixture with serum, constitutes the so-called tuberculous pus. In rare instances a focus of softening is replaced by fibrous tissue, constituting "healing by sclerosis."

When a tuberculous focus is established in the lung its tendency is almost invariably toward progressive extension. When the bronchial tubes are involved, a tuberculous chronic bronchitis ensues, and infective material is conveyed to adjacent portions of the lung by the propulsive action of the inspired and expired air during respiration. The infection also progresses by continuity of the pulmonary tissues, the infective focus progressively involving adjacent lobules of the lung. The infective organism is also disseminated by the medium of the pulmonary lymphatics and by the blood stream. During the evolution of the tubercle a vein is apt to be eroded and the bacilli widely disseminated throughout the lung and indeed throughout the general circulation. Infection of an entire lung is usually explained in this way.

The initial lesion of pulmonary tuberculosis develops in the vast majority of cases in the apical portion of the lung. The apparent predilection of the tubercle bacillus for this portion of the lung is probably due to the fact that the apices of the lungs are less mobile than are the bases, resulting in a relative retardation of the flow of blood and lymph in this region, which favors infection with the tubercle bacillus.

Pulmonary tuberculosis occurs in four forms; namely, *acute miliary tuberculosis; acute tuberculo-pneumonic phthisis*, of which there are two varieties, the *pneumonic*, and the *bronchopneumonic; chronic ulcerative phthisis;* and *fibroid phthisis.*

ACUTE MILIARY TUBERCULOSIS OF THE LUNGS

Clinical Pathology.—This form of pulmonary tuberculosis results from the erosion of a vein by a tuberculous focus, whereupon the bacilli are distributed throughout the lungs and by way of the blood stream to various organs of the body. The disease has not infrequently followed attacks of pertussis and measles during childhood. For a time following the erosion of the vessel and the dissemination of the organisms through the body the subject of the disease presents the picture of an acute generalized infection with hepatic and splenic enlargement. After a variable period the symptoms and signs become localized in the bronchopulmonary system, and the lungs upon autopsy are found studded with innumerable miliary tubercles, while the bronchi and bronchioles are the seat of a tuberculous bronchitis.

Physical Signs.—*Inspection.*—The physical signs of acute miliary tuberculosis of the lungs are in the main those of acute catarrhal bronchitis, with, however, a more extreme grade of dyspnea, cyanosis developing upon very slight exertion. Cough is persistent, and is attended by the raising of mucopurulent sputum, which early in the course of the disease becomes sanguineous, with the coincident presence of tubercle bacilli.

Palpation.—Palpation of the thorax in acute miliary tuberculosis seldom yields any diagnostic data. The pulse is unduly accelerated from the onset of the disease, frequently exceeding 160 beats per minute. Palpation of the spleen reveals enlargement of that organ.

Percussion.—In acute miliary tuberculosis of the lungs the findings upon percussion of the thorax are variable. As a rule, the note is moderately hyperresonant, despite the wide dissemination of miliary tubercles throughout the lungs. In the case of children, in whom the disease has arisen as a sequela of measles or pertussis, the percussion findings are very similar to those of nontuberculous bronchopneumonia, with scattered areas of dullness over the bases and Skodaic resonance in the infraclavicular and mammary regions. In cases which are attended by pleural effusion dullness or flatness is elicited over the dependent portions of the lungs.

Auscultation.—Moist râles are demonstrable over both lungs, becoming more numerous and assuming the mucous type toward the termination of the disease. Pleural friction is occasionally encountered; and Jürgensen has described a fine crepitation which caused by the presence of miliary tubercles upon the pleura. In children the breath sounds over the bases posteriorly are often bronchovesicular or even frankly bronchial in type.

Diagnosis.—The extreme degree of dyspnea and cyanosis upon the slightest exertion serve to place the disease above the signs of a simple catarrhal bronchitis of nontuberculous origin. In children it is difficult for a time to differentiate the disease from a nontuberculous bronchopneumonia. When, however, the sputum becomes sanguineous and when tubercle bacilli are detected in the expectoration, the differential diagnosis is plain. In the adult subject the progressive downward course of the disease and the failure of the fever to terminate by crisis serve to eliminate the possibility of lobar pneumonia.

ACUTE PNEUMONIC PHTHISIS

Clinical Pathology.—In acute pneumonic phthisis the disease involves the greater portion of a lobe, or indeed an entire lung, the morbid process having its inception in a tuberculous focus in one apex. This form of phthisis attacks males with greater frequency than females, and the subjects of the disease frequently have been in robust physical condition until the inception of the disease. Exposure to the elements is not infrequently the immediate precursor to the onset of the acute symptoms, while in other instances the disease develops during convalescence from an attack of influenza or other acute infectious disease.

During the early stage of the disease the alveoli are the seat of dense consolidation which resembles that of lobar pneumonia during the stage of hepatization. In the further evolution of the disease, excavation of the lung ensues with cavity formation. The pleura corresponding to the area of pulmonary disease is inflamed, and is clothed with a variable amount of fibrinous exudate.

The sputum, which is at first scanty and mucoid, becomes abundant and serosanguineous, containing elastic fibers and tubercle bacilli. The latter are occasionally demonstrable in the sputum during the first week of the disease.

Physical Signs.—The physical signs of acute pneumonic phthisis correspond in the main to those of lobar pneumonia, with certain differences in the area of distribution of the principal signs of the diseases.

Inspection.—Respiration is rapid and labored, and occasi
the disease is attended by repeated attacks of imminent su

with cyanosis. The cheeks commonly wear a hectic flush; an
herpes labialis, which is so frequently observed in lobar pneumonia
is only occasionally noted in acute pneumonic phthisis.

Palpation.—Vocal fremitus is exaggerated over the consolidate
area, which usually occupies the apex or upper lobe of one or bot

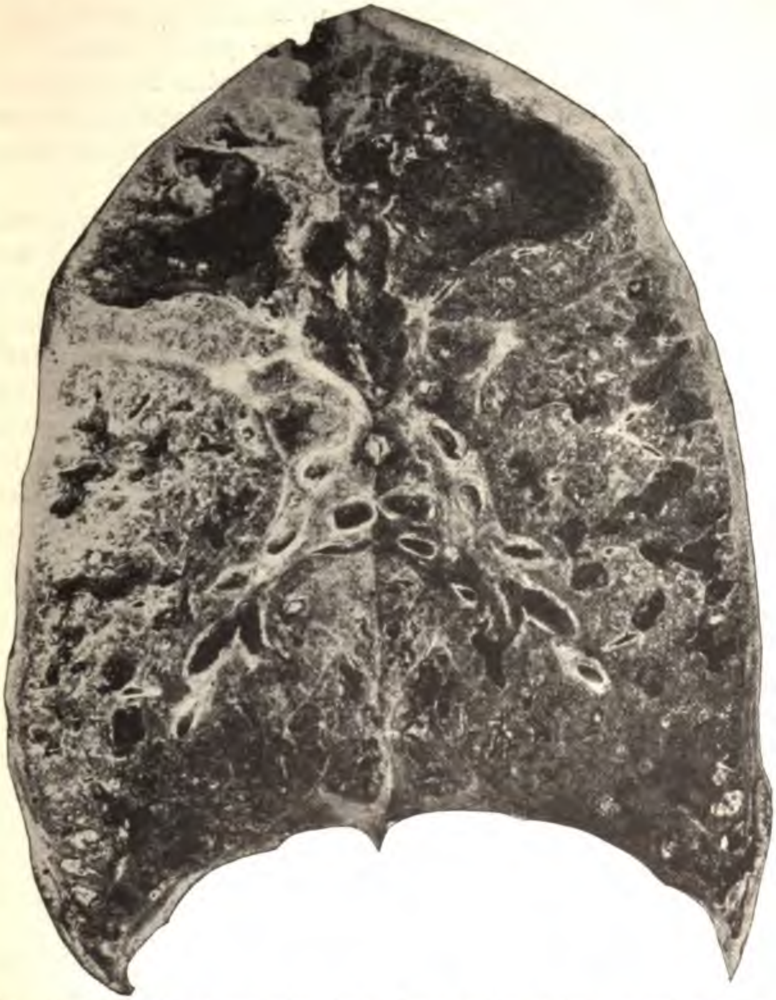


Fig. 108.—Illustrating caseous tuberculosis. Large cavities at the apex and many sma
cavities throughout the lung. (Pottenger, after Tendeloo.)

lungs. Palpation of the supraclavicular regions during inspiratio
deficient inspiratory expansion of the apices of the lung
rcussion of the thorax during the first few day

of the disease reveals normal resonance, or indeed, a slightly hyperresonant note, which early is succeeded by dullness due to consolidation. Late in the course of the disease, after cavitation has supervened, the note changes again and it is possible in many instances to elicit one or more of the classical signs of cavity.

Auscultation.—The earliest auscultatory findings are a partial suppression of the vesicular murmur, which later in the evolution of the disease becomes bronchovesicular or purely bronchial in character. This purely tubular breathing persists for a week or ten days, when, instead of clearing and disappearing as is the case in lobar pneumonia, signs of cavity formation, indicating softening and excavation of the lung, supervene.

Diagnosis.—Acute pneumonic phthisis must be differentiated from lobar pneumonia, which it closely resembles during the early stages. However, it is to be remembered that phthisis is prone to have its inception in the apices while lobar pneumonia is prone to involve the base of the lung. Moreover, at the seventh or ninth day, instead of terminating by crisis with amelioration of the more acute symptoms, the disease progresses and pursues an aggravated course, with sweats, and the appearance of elastic fibers and tubercle bacilli in the sputum. Moreover, in the case of phthisis the opposite lung is apt to yield similar physical signs, though to a minor degree; and it is occasionally possible to obtain a history of previous tuberculosis in the subject.

ACUTE BRONCHOPNEUMONIC PHTHISIS

Clinical Pathology.—Acute bronchopneumonic phthisis, "phthisis florida," or "galloping consumption," is most frequently observed in children, in whom it arises as an independent affection, but with far greater frequency as a sequela of one of the acute infections, notably after measles and pertussis. With less frequency the disease attacks adults, and not infrequently those who are in the prime of health.

The disease is a caseous bronchopneumonia, with its point of inception in the terminal bronchioles, which early become occluded by a caseous material, while the alveoli are filled with the products of an acute catarrhal pneumonia. The areas of consolidation are as a rule widely disseminated throughout the upper lobes of the lungs. By fusion of adjacent areas of consolidation, almost an entire lobe may become airless; but in most instances the patches of consolidation are separated by bands of crepitant pulmonary tissue.

Physical Signs.—*Inspection.*—The subject of bronchopneumonic phthisis is liable to attacks of extreme dyspnea and cyanosis, with cough, progressive emaciation, and hectic flushing of the cheeks. As the disease progresses, the patient becomes dull and somnolent, with dry lips and tongue, and a general appearance of torpor.

Palpation.—Palpation frequently reveals exaggeration of vocal fremitus in the infraclavicular and mammary regions, and palpation of the supraclavicular fossæ shows lagging expansion of the apices during inspiration.

Percussion.—The note upon percussion is seldom frankly dull. Rather it has a vesiculotympanitic quality, owing to the presence of multiple patches of consolidation separated by areas of crepitant lung.

Auscultation.—The respiratory murmur over the areas of consolidation is bronchovesicular, not attaining to the purely bronchial type, with crepitant and subcrepitant râles. Late in the disease, with the supervention of softening and cavitation, amphoric or cavernous breathing is apt to be encountered.

Diagnosis.—Acute bronchopneumonic phthisis must be differentiated from nontuberculous bronchopneumonia, a problem which requires time and observation of the patient. A bronchopneumonia which has its inception in the apices is strongly suggestive of phthisis. The rapid emaciation of the subject points to the same origin of the disease; while with the detection of signs of pulmonary excavation and with the advent of the tubercle bacillus in the sputum, the diagnosis is assured.

CHRONIC ULCERATIVE PHTHISIS

Clinical Pathology.—Chronic ulcerative phthisis has its inception in a tuberculous focus in one or both apices and the disease extends progressively downward, involving lobule after lobule, and lobe after lobe of the lung. From an apical lesion infective material is aspirated into the bronchial tubes of uninfected portions of the lung and here sets up tubercle formation about the finer bronchioles. Thence the disease spreads to the infundibula, and with less frequency, ascending infection occurs, leading to infection of the bronchi immediately above the smaller bronchioles.

The infective process also travels by continuity of tissue from a primary focus to the immediately surrounding portions of the lung. Infection frequently travels by the lymphatics or the blood stream, infecting other portions of the lung. Through these avenues the infection of the lung when once established, progressively attacks the several portions of the lung.

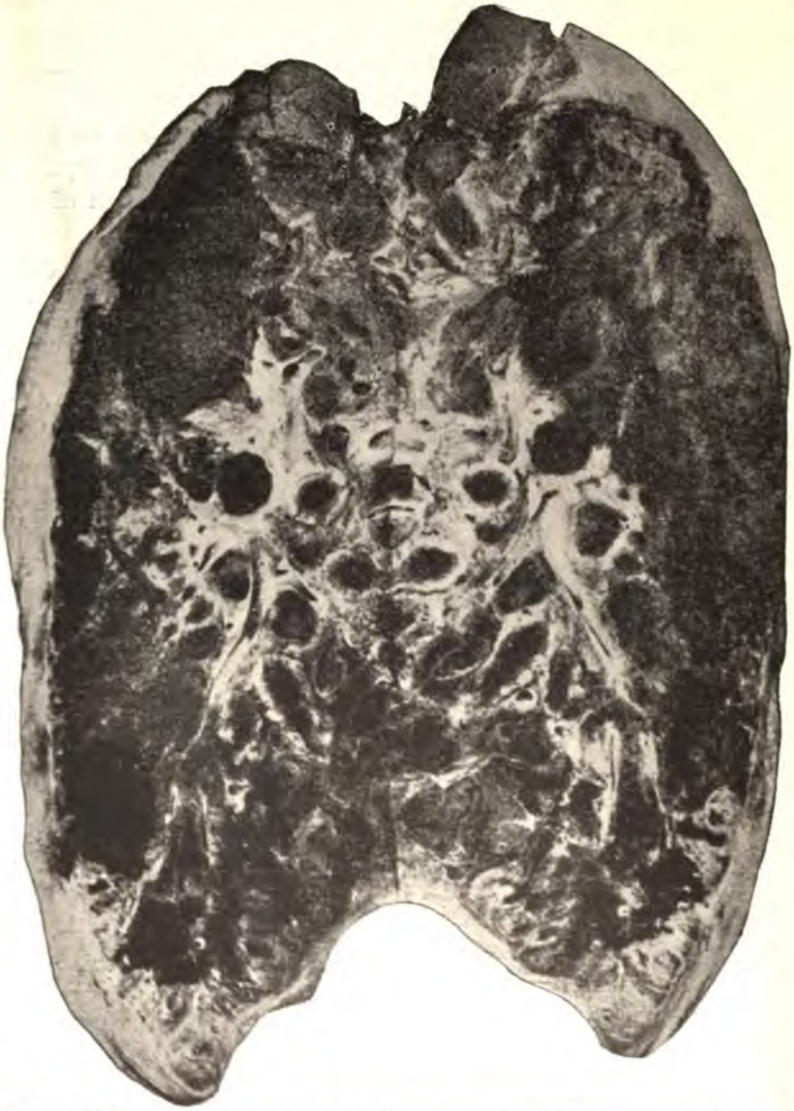


Fig. 109.—Illustrating pulmonary tuberculosis, with thickened pleura, many bronchiectatic cavities, and generalized cavity formation. (Pottenger, after Tendeloo.)

In the further evolution of the disease different portions of the lung show tubercles in different stages of infiltration, caseation, or softening, leading eventually to cavity formation. Ulceration of the walls of the bronchial tubes not infrequently permits stretching of these walls during paroxysms of cough or from the weight of stagnant secretion, with the formation of bronchiectatic dilatations.

Aside from the bronchiectatic cavities, excavation of the lung occurs apart from the bronchi. The walls of these cavities in certain instances are smooth, while in other cases they are uneven and rugged. In these cavities blood vessels which have not been destroyed may be found traversing the cavities; and by rupture they may produce copious hemorrhage, which may prove fatal. Cavities of moderate size by coalescence often lead to the formation of extensive excavations, which, in exceptional instances may embrace the greater portion of a lobe of the lung. The cavities of chronic ulcerative phthisis occur with great frequency in the upper lobe of the lung, whereas the cavity which is of bronchiectatic origin is commonly situated in the lower lobe.

When the peripheral portions of the lung are involved, a cavity is apt to form immediately subjacent to the pleura, and by rupture through that membrane is apt to produce pneumothorax. In other instances, instead of rupturing with the formation of a fistulous communication between the lung and the pleural cavity, adhesions may form between the visceral and the parietal pleura as the result of localized pleurisy overlying the tuberculous lesion of the lung. These pleural adhesions are frequently quite extensive, to a great extent serving to immobilize the lung.

Instead of undergoing caseation and softening with consequent cavity formation, tuberculous foci in the lung may undergo a process of sclerosis. Sclerosis is a reparative process, tending to inhibit the spread of infection; but it is uncommon for sclerosis to occur to an extent which is sufficient to save a tuberculous lung. Lime salts may be deposited in sclerotic and caseous foci and limit the spread of the infection temporarily; but an attack of a bronchial affection such as influenza is prone to "light up" these dormant or residual foci of infection.

The bronchial glands do not escape the tuberculous infection. Infiltration, caseation, abscess formation, and rupture of the glands are frequently encountered.

Physical Signs.—The physical manifestations of chronic ulcerative phthisis are variable, depending upon the duration and the progress of the individual case. All gradations are encountered, A more frequent anatomic change consists in moderate flattenin of the thorax, together with supraclavicular depressions of un equal depth. Also there is lagging expansion at one apex, whic is often so slight as to require palpation of the supraclavicula area for its detection. Moderate pallor of the skin and mucou

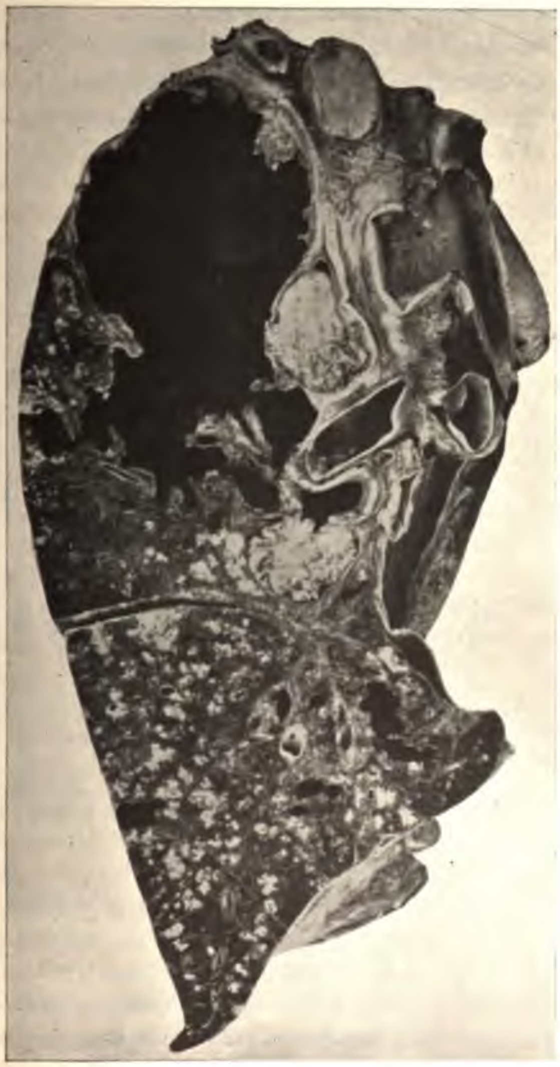


Fig. 111.—Lung. Chronic phthisis, showing a large irregular cavity in the upper lobe In the lower lobe there are scattered acute nodules grouped in clusters around the smal bronchi; and also several small more acute cavities. The bronchial glands are enlarge and caseous. (Edinburgh University Anatomical Museum.) (Woolley after Beattie an Dickson.)

membranes is often in evidence. Many subjects of incipient phthisis exhibit an abnormal flattening and mobility of the sternal angle (Rothschild's sign); while in other cases there is early ankylosis and preternatural rigidity of the vertebral column in the thoracic region (Lorenz's sign).

In advanced cases of chronic phthisis the phthisical or alar thorax is in evidence, with undue prominence of the bony thorax and atrophy of the soft structures. Unilateral deficiency of expansion is noted, most markedly in the infraclavicular and mammary regions. In the very late case the thorax frequently exhibits bilateral deficiency of expansion, the principal respiratory movement of the thorax occurring in a vertical direction. In the presence of advanced tuberculosis of the left lung the heart exhibits a wide impulse in the third and fourth interspaces. In not a few instances the pupil corresponding to the side of the principal pulmonary disorganization is dilated as a result of pressure exerted upon the cervical sympathetic fibers. In cases of extreme unilateral disease the vertebral column is bowed with t
concavity directed toward the side of the disease. Local retra
of the chest wall is frequently to be noted, indicating area

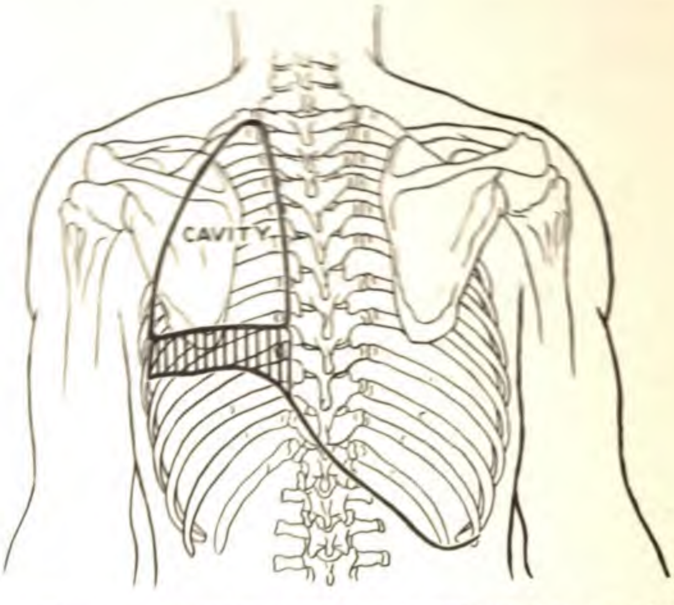


Fig. 112. Illustrating compensatory change in right lung with depression of the diaphragm following extensive cavitation of left lung.

monary collapse or the traction of pleural adhesions. The finge are often Hippocratic, presenting clubbing of the terminal ph langes.

As the disease progresses, dyspnea becomes a prominent featu of the picture, partially as a result of pulmonary excavation, ar partially as a consequence of the development of emphysem and late in the course of the disease as a manifestation of rig heart failure. Coincidentally the pallor of the skin and muco membranes increases; the skin of the thorax is apt to prese patches of yellowish-brown pigmentation; and, as the right hea fails, edema of the feet and ankles develops rapidly.

The sputum is characteristic of the disease. Scanty and almo purely mucoid in the early stages of the disease, later the sputu becomes abundant, mucopurulent, or purulent, containing leuc cytes, epithelial cells, elastic fibers, tubercle bacilli, and vario associated bacteria. Small yellowish caseous masses are fr quently found in the sputum, which are most intimately asso ated with the tubercle bacillus. Occasionally the sputum blood-tinged or contains free blood. Hemoptysis is a valuab sign of the disease.

Palpation.—In incipient phthisis palpation of the apices is a to reveal the presence of deficient expansion at one apex. In apic consolidation palpation of the infraclavicular region yields i creased vocal fremitus. In view of the fact that vocal fremitus normally more intense over the right apex than it is over the le an equalization of the fremitus upon the two sides points to co solidation of the left apex.

In advanced phthisis, over densely infiltrated or consolidat areas of the lung vocal fremitus is exaggerated. The fremit exhibits the acme of exaggeration over a superficial cavity wi patent bronchial communication. Extensive pleural thickenin pleural exudate, or pleural effusion effectually masks or abolish the vocal fremitus. With less constancy it is possible to eli friction fremitus or rhonchal fremitus upon palpation of the thor over an area of consolidation.

Early in the course of the disease the pulse is moderately i creased in rate, but not out of proportion to the fever, and i volume is maintained. In the further evolution of the disease t frequency is increased with diminution in the volume combin with arrhythmia of the pulse.

[illegible]ussio[illegible] [illegible] [illegible]cipient phthisis percussion of the apices [illegible] [illegible]fraclavicular, suprascapular, and upp

show the tubercle bacillus. Moreover, there is frequently a history of tuberculosis in one of the antecedents.

Consolidations of the lung due to lobar or lobular pneumonia are differentiated from that of tuberculosis by the different clinical courses of these diseases and by the absence of the tubercle bacillus from the sputum.

Bronchiectasis, pulmonary abscess, and *pulmonary gangrene,* while productive of physical signs suggestive of phthisis, are differentiated by the distribution of the signs and the absence of tubercle bacilli from the sputum.

FIBROID PHTHISIS

Clinical Pathology.—Fibroid phthisis is a form of chronic pulmonary tuberculosis, in the evolution of which the predominant feature is the formation of an excessive amount of fibrous connective tissue. In the incidence of the disease the tuberculous infection of the lung may be the primary affection, and through the process of reparative sclerosis the disease may assume the fibroid type. On the contrary, the tuberculous infection may be engrafted upon a lung which was previously fibrosed, either as a result of pneumonokoniosis or chronic interstitial pneumonia following an unresolved lobar pneumonia which has healed by sclerosis, or a pleurogenous interstitial pneumonia.

In the cases which are primarily tuberculous, the disease has its inception in an apex of one or both lungs; and, as in other forms of the disease, it progresses downward, involving the lower lobes in regular progression. In this class of cases one apex may be sclerosed, or the entire lung may be sclerotic and shrunken, showing cavities and bronchial dilatations, which may be empty or which may be filled with caseous material. In many instances the only way of determining whether a pulmonary fibrosis is tuberculous or nontuberculous is by the presence or absence of the tubercle bacillus in the sputum during the life of the patient.

The distribution of the fibrosis is largely influenced by the manner of production of the disease. In cases which arise from inhalation through the respiratory passages, the formation of connective tissue assumes the character of a peribronchial fibrosis, and is most pronounced around the bronchi and bronchioles; whereas in the cases which develop as a sequela of tuberculous pleurisy the peripheral portions of the lung are most ex

involved and bands of fibrous connective tissue pass from t
pleura into the deeper portions of the lung.

Whatever the mode of production of the disease, the ultima
result is a marked diminution in the size of the lung which
shrunken, pigmented, and occupies a very small area of the upp
portion of the pleural cavity near the vertebral column.

Physical Signs.—*Inspection.*—The physical findings in fibro
phthisis are almost identical with those of chronic interstit
pneumonia. The side of the thorax corresponding to the fibros
lung is shrunken, presenting local retractions, with droopi
shoulder, and a minimal degree of expansion in the advanced ca
of the disease. Scoliosis is frequently present with the thora
deformity, with the concavity directed toward the side of the d
ease. Likewise, the cardiac impulse is displaced toward the si
of the cirrhotic lung. In left-sided disease there is frequently
wide impulse in the third and fourth interspaces; while in disea
of the right lung the impulse is frequently invisible, as it is d
placed toward the right to such degree as to lie behind t
sternum.

The intercostal spaces are narrowed, and the ribs occasiona
overlap upon the side of the diseased lung. The depressions
the supraclavicular and infraclavicular regions are abnorma
deep, and the clavicles are unduly prominent. The sound side
the thorax presents vicarious expansion as a result of compensato
emphysema of the sound lung.

Palpation.—In incipient cases palpation is serviceable in dete
ing slight degrees of deficient expansion which have escap
detection during inspection of the thorax. Vocal fremitus var
in intensity with the condition of the lung and the pleura. P
pation of the thorax overlying a cirrhotic lung which is imn
diately subjacent to the chest wall and palpation over pulmona
cavities yield exaggeration of vocal fremitus; whereas in t
presence of excessive pleural thickening, and when the lung
shrunken and is not in contact with the thoracic parieties, vo
fremitus is diminished in intensity or is abolished. Similar
when the fibrosis is centrally situated and is covered by norn
pulmonary tissue, vocal fremitus is unaltered or is actually dim
is [illegible] palpation of the thorax over the sound lung,
[illegible], vo[illegible]us is diminished as a result of ra
[illegible]nsatory emphysema.

[illegible] apex upon the diseased s
[illegible]sonance owing to retraction

the pulmonary apex. Cavitation in the upper lobe of the lung is indicated by hyperresonance or tympany in the infraclavicular region. In cases with excessive pleural thickening the percussion note is impaired and there is a marked sense of resistance as appreciated by the pleximeter finger. Tympany over the lower lobe of the lung is indicative of cavitation which is commonly bronchiectatic in origin. Percussion of the sound lung in unilateral disease yields hyperresonance.

Auscultation.—The respiration over the upper lobe of the lung in the infraclavicular region is commonly bronchovesicular or frankly bronchial; and in the presence of cavitation in this region is amphoric or cavernous. At the bases there is frequently distinct bronchial breathing, unless the disease be so advanced that the lung is retracted into the upper portion of the pleural cavity, in which event the respiratory murmur is distant or entirely inaudible. Râles of chronic bronchitis are quite constantly present over the diseased lung. The pulmonic second sound is apt to show accentuation.

Diagnosis.—Deformity of the thorax, the wide area of the cardiac impulse in left-sided disease and absence of the impulse very frequently in right-sided disease, the bronchial or amphoric breath sounds over an apex of the lung, associated with dullness or tympany as the case may prove, indicate fibrosis of the lung with cavity formation. Whether or not this is tuberculous in origin is to be determined by the examination of the sputum for the tubercle bacillus. But in fibroid phthisis, in contradistinction from the fibrosis of chronic interstitial pneumonia, the disease is apt to be bilateral, whereas in the case of fibrosis from chronic interstitial pneumonia it is apt to be unilateral. It follows that careful examination of both lungs should be practiced for the purpose of detecting signs of disease in an apparently normal lung.

PULMONARY SYPHILIS

Clinical Pathology.—Syphilis attacks the lung in two forms; namely, as *congenital syphilis,* and as *acquired syphilis* of the lung.

Congenital syphilis of the lung was first described by Virchow as "pneumonia alba." The lung in congenital syphilis is enlarged, showing on its external surface indentations corresponding with the ribs with which it is in contact. The lung is white or slightly tinged with yellow; it is firm; and upon section the cut surface resembles

macroscopically a section of pancreatic tissue, a condition to whi
Lorain and Robin applied the term "pancreatization."

Microscopically the interalveolar septa show an overgrowth
fibrous connective tissue, leading to thickening of the alveolar wal
the alveolar spaces being smaller than normal and densely pack
with desquamated epithelial cells, cellular detritus, and fat. Ho
man called attention to a thickening of the vascular walls in t
interalveolar septa, analogous to that which occurs in syphilit
fetal tissue elsewhere, the vessels often presenting, as well, e
dences of hyaline degeneration.

The lesions of acquired syphilis of the lung may assume thr
types; namely, gummata, interstitial sclerosis analogous to chron

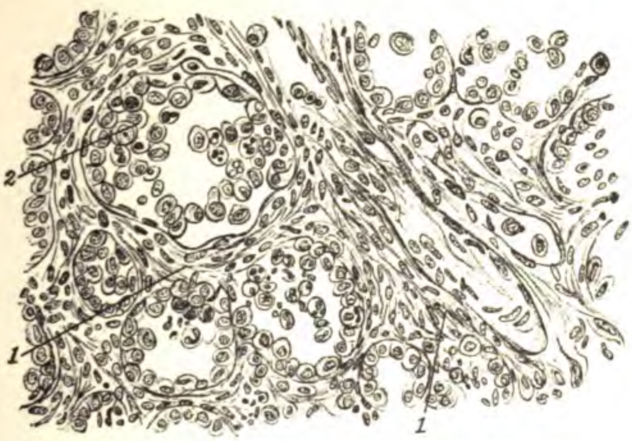


Fig. 113.—Pneumonia alba of newborn. (From McFarland.)

interstitial pneumonia from other causes, and syphilitic bronch
pneumonia.

The gummata are situated deeply near the root of the lung, var
ing in size from a hazelnut to a hen's egg or larger. They are pro
to soften and to break into a bronchus or to undergo sclerosis ar
by traction lead to bronchiectatic dilatations. Gummata are t
rarest of syphilitic lesions of the lungs. Wagner and Henop d
scribed gummata in both the upper and lower lobes of the lun
situated principally toward the root, usually containing in the
center a dilated bronchus with chronically inflamed mucous mer
brane. The lung intervening between the gummata was partial
crepitant, while the apices and anterior borders of the lungs we
in a state of compensatory emphysema.

The interstitial sclerosis attending acquired syphilis of the lu has its inception near the root of the lung, and extends thence in various directions between the lobules of the lung. The patches of insular sclerosis often extend in all directions from gummata situated near the pulmonary root, dividing the lung into a number of artificial subdivisions. Traction diverticula are formed, which lead to bronchiectases.

The bronchopneumonia of syphilis does not differ essentially from bronchopneumonia of other causes, Pavloff describing a bilateral, syphilitic bronchopneumonia with patches of consolidation interspersed between areas of normal tissue in both lungs. The alveoli are filled with desquamated epithelial cells, with a variable number of erythrocytes and leucocytes, the pneumonic patches being dull slate-colored upon section.

Physical Signs.—Congenital syphilis of the lung must in certain instances be differentiated from atelectasis, which it closely simulates; but there are usually sufficient evidences of congenital syphilis upon the exterior of the body to render the diagnosis clear.

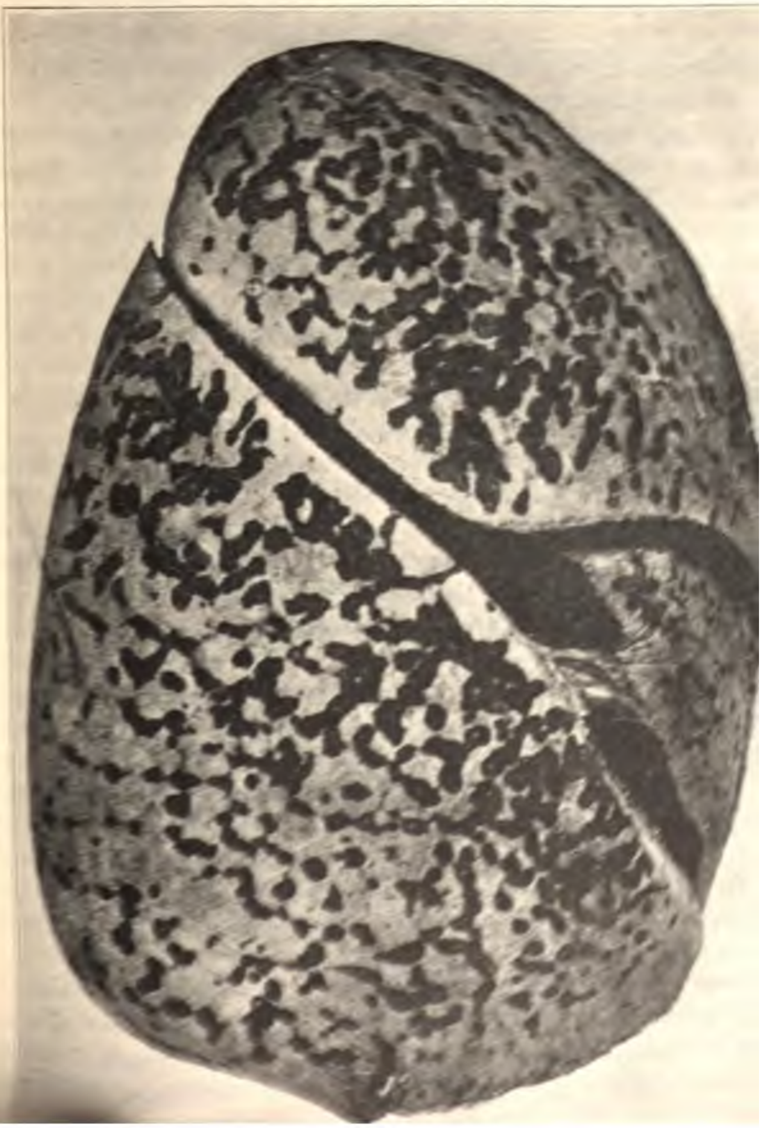
The physical signs of acquired syphilis are not characteristic, the clinical picture being often that of chronic interstitial pneumonia, from other causes, chronic ulcerative phthisis, or ordinary bronchopneumonia. However, there are certain localizations of the principal signs emanating from pulmonary syphilis which are of considerable aid in diagnosis. Thus, the lesions are usually situated near the root of the lung, gummata and sclerosis in this situation giving rise to dullness upon percussion and bronchial breath sounds upon auscultation along the lateral sternal borders, over the roots of the lungs, which signs decrease in intensity as the examiner progresses outward, upward, and downward from this area. Grandidier lays great emphasis upon the localization of the physical signs in these regions of the thorax, while Pankritius points out the importance of dullness in the interscapular regions at the same level.

The disease is productive of chronic cough, which is not infrequently attended by hemoptysis and fever, simulating rather closely the picture of chronic ulcerative phthisis; but there can often be found evidences of syphilis in other regions of the body, and the specific tests for syphilis are available.

PNEUMONOKONIOSIS

Clinical Pathology.—Pneumonokoniosis is a chronic in
of the lungs due to the inhalation of various dusts a

particles. Depending upon the nature of the exciting cause, t
disease is subdivided into several different types: *siderosis*, fr
the inhalation of iron filings; *chalicosis*, from the inhalation
stone particles; and *anthracosis*, from the inhalation of coal du
Similar pulmonary changes ensue upon the continued inhalation



the fibers of wool, flax, cotton, tobacco, or particles of glass, bone, or horn, in the course of an occupation.

Anthracosis, or "coal miner's disease," arises as the result of the prolonged inhalation of fine particles of coal. The minute amounts of this dust which are usually inhaled by persons who do not pursue an occupation which habitually brings them into contact with coal dust are absorbed by the leucocytes which are present upon the surfaces of the respiratory passages; and they are carried upward by the action of the ciliated epithelium of the respiratory tract, and are expectorated. When, however, the dust is inhaled in larger amount, some of the coal particles penetrate the bronchial mucous membrane and find lodgment in the subjacent connective tissue layer, or enter the lymph stream and are conveyed to the smaller lymphatic glands around the blood vessels, the bronchi, the pleura, or in the mediastinum. The lungs of all dwellers in cities are moderately pigmented from the inhalation of coal dust and soot, while the lungs of persons who have lived all their days in the open country, remote from large manufacturing industries, are frequently pink in color and free from this pigmentation.

When soot or coal dust is inhaled in excessive quantities, as in the case of the coal miner, a portion of it penetrates to the ultimate ramifications of the bronchioles and to the pulmonary alveoli. In the lungs which are the seat of this extreme grade of anthracosis the organs are distinctly black. The irritation of the fine grains of dust in the interstices of the pulmonary tissues excites the proliferation of fibrous connective tissue, resulting in fibrosis in insular foci. Upon section these fibrosed areas are hard to the touch and they exude a black fluid. Diffuse induration of an entire lung, or of the greater portion of a lung, is commonly encountered at autopsy.

The bronchial and mediastinal lymphatic glands share in the general induration, and they are frequently the seat of periadenitis by virtue of which they adhere to the adjacent large vessels, and by rupture into the same they distribute the pigmentation to various organs of the body, as the liver, spleen, kidneys, and mesenteric lymphatic glands.

Bronchial perilymphadenitis may lead to the formation of adhesions between the glands and the pericardium, producing a chronic mediastinopericarditis. Adhesions are apt to form between these glands and the esophagus, and lead to the formation of esophageal diverticulum. Similarly adhesions are prone to form between the mesenteric glands and the aorta, with subsequent erosion of that vessel with fatal hemorrhage. Adhesive bands ma strict

the trachea or adjacent blood vessels, producing tracheal steno
with stridulous respiration, or vascular constriction with the p
duction of murmurs. Adhesions may produce aspiration pne
monia when a gland erodes a bronchus and discharges its conte
by that avenue. Moreover, the chronically inflamed glands m
produce vagus or recurrent laryngeal nerve paralysis by pressu
upon these nerves.

Chalicosis, due to the inhalation of the fine dust of alumi
quartz, or sandstone, is known as "stone-cutter's phthisis," mi
stone maker's phthisis," "grinders rot," or "potter's asthma
Chalicosis produces a greater degree of induration of the lung th
does any other form of pneumonokoniosis.

Siderosis is a fibrosis of the lung due to the inhalation of ir
filings or iron dust. The changes in the lungs and the mediasti
and bronchial glands are similar to those which accompany anth
cosis; but the induration attains a greater degree than it does
anthracosis.

The areas of insular sclerosis which are formed throughout t
lungs, in certain cases undergo softening and form pulmona
cavities. In other instances the softening is due to subsequent i
fection with the tubercle bacillus; although, as a rule, the pne
monokoniotic lung does not appear to prove a fertile field for t
ravages of the tubercle bacillus. As a result of the chronic infla
mation which is induced by the continual aspiration of the irrita
dusts, the bronchial tubes show a chronic bronchitis, which is f
lowed in many cases by emphysema, while the lung slowly und
goes a slow, insular sclerosis.

Physical Signs.—The physical signs of pneumonokoniosis a
modified and influenced by the coincident chronic bronchit
emphysema, or interstitial sclerosis, with occasional signs
excavation of the lung engrafted upon these signs. Signs
cavity suggest chronic ulcerative phthisis or bronchiectasis, a
it should be borne in mind that both diseases are possible comp
cations of pneumonokoniosis. In a typical case of pneumon
koniosis the physical signs are evolved with a fair degree
regularity. The earliest signs to become manifest are those of
chronic bronchitis; then there appears the picture of a gradual
developing hypertrophic emphysema; and finally signs of chron
interstitial pneumonia become evident, with or without signs
bronchiectasis or cavitation.

The sputum in these cases is frequently of assistance in esta
lishing the diagnosis. In anthracosis it is black from the conte

of coal dust; in siderosis it is reddish or brown; while in chalicosis the shining particles of stone dust can be seen microscopically.

Diagnosis.—The diagnosis is to be founded upon a history of an occupation which requires the long-continued inhalation of dusts, and upon physical signs of chronic bronchitis, emphysema, and insular sclerosis of the lung, with or without evidences of cavity formation, and upon the characteristic sputum. Late in the course of the disease, the sputum in a suspected case may show the tubercle bacillus from subsequent tuberculous infection.

ATELECTASIS

Clinical Pathology.—Imperfect expansion of the lung or partial collapse of a lung which has become fully expanded may be congenital or acquired. In the congenital form of atelectasis the lung has never attained its proper degree of expansion; while in the acquired form, which is a disease of later life, there occurs from various causes a partial collapse of the lung.

Congenital atelectasis is a disease of the newly born, developing usually as a result of insufficient inflation of the lung due to the aspiration of meconium or mucus during parturition, or from abnormal weakness of the respiratory muscles subsequent to birth. In atelectatic children who survive, the anterior borders and the apices of the lungs are partially inflated, while the central and lower portions of the lungs are brownish red, vascular, and fail to crepitate upon manipulation.

As the child gains strength the anterior and upper portions of the lungs become emphysematous from compensatory emphysema, while the deeper portions are very tardy in attaining their proper degree of expansion. Congenital atelectasis is a bilateral disease, affecting both lungs, and usually to a similar degree. It is probable that the central portions of the lungs of atelectatic children never attain to full inflation, as the tendency is rather for secondary changes to occur, which lead to sclerosis and contraction of the deeper portion of the lung near the pulmonary root.

Acquired atelectasis is frequently the result of compression of the lung by a pleural effusion, a tumor, aneurysm, or deformity of the thoracic wall. The most fruitful source of the disease, however, is bronchial obstruction from foreign bodies, or external pressure from a pulmonary tumor or aortic aneurysm. Similarly, in the course of capillary bronchitis or bronchopneumonia the terminal bronchioles are obstructed by inflammatory exudate, leading to circumscribed areas of atelectasis. Conditions of great debility, the result of malnutrition or lying long in the recumbent posture with an exhausting disease, occasionally induce areas of circumscribed atelectasis.

The atelectatic area of the lung is darker than the adjacent tissues, is depressed below the surface of adjacent areas of the lung, and is usually distributed in a number of areas corresponding to lobules of the lung. Upon section the areas are usually dry, but they may be moist from the presence of chronic passive congestion.

Physical Signs.—The physical signs of atelectasis are influenced and modified by the primary manifestations of the disease or condition which has been the occasion of the atelectatic state of the pulmonary tissues. The signs also vary in intensity with the volume of lung involved in the atelectasis and the condition of the undiseased portion of the bronchopulmonary system. It is obvious that the physical signs arising from collapse of an entire lobe or an entire lung will differ markedly in distribution and degree from those which are referable to a few scattered areas of pulmonary collapse.

Inspection.—Extensive atelectasis, involving a considerable area of a lung, gives rise to dyspnea and occasionally to cyanosis, with inspiratory retraction of the lower intercostal spaces and the epigastrium. In congenital atelectasis the child suffers with repeated suffocative attacks during which immediate dissolution seems to be imminent. The expansion of the thorax is materially diminished in extensive atelectasis.

Palpation.—Vocal fremitus over the area of disease may be diminished, absent, or exaggerated, depending upon the state of the pulmonary parenchyma. Collapsed, toneless pulmonary tissue fails to conduct the vocal vibrations with the normal intensity; and if a main bronchus is completely obstructed, there is absence of vocal fremitus over the distribution of the bronchus. However, when secondary changes have been established in a case of atelectasis with partial bronchostenosis, the fremitus is transmitted by the consolidated or sclerotic tissues with undue intensity to the thoracic wall. Pleural friction fremitus is occasionally demonstrable, due to involvement of the pleura over the atelectatic area.

Percussion.—The dullness which would be produced by small patches of atelectasis is effectually masked by the hyperresonance of the adjacent emphysematous areas. In order to afford dullness upon percussion an area of atelectasis must be large and situated in the peripheral portion of the lung. A patch which directly overlies a large bronchus yields the tympany of the bronchus upon forcible percussion. Deeply seated areas of atelectasis fail to yield dullness, owing to the intervention of the normal crepitant tissues between the area of disease and the chest wall.

Auscultation.—The vesicular murmur is feeble or abolished over an area of atelectasis, unless the area of collapse overlies a large bronchus, in which event the murmur is bronchovesicular or frankly bronchial. In the presence of nonextensive areas of collapse the only auscultatory sign may be the presence of a few crepitant râles upon full inspiration. This holds true of the cases of atelectasis which develop in the dependent portions of the lungs in patients who have been long in the recumbent posture with exhaustive disease. In cases of extensive atelectasis the second sound of the heart is accentuated at the pulmonary area.

Diagnosis.—The diagnosis of pulmonary atelectasis is frequently for a time difficult or impossible. The finding of respiratory embarrassment and the location of some adequate causative lesion such as bronchial obstruction or pulmonary compression is suggestive. The physical signs are seldom definite and distinctive, as small areas of collapse have their physical signs effectually masked by the emphysematous state of the surrounding lung. Moreover, as a larger area of collapse is apt to overlie a large bronchus and to have its tympanitic note engrafted upon the dullness of the atelectasis, a pulmonary cavity is apt to be suspected where none exists. The fact that the physical signs have a tendency to improve and to regress with changes of posture and with deep inflation of the lungs is suggestive of atelectasis.

HYPERTROPHIC EMPHYSEMA

Clinical Pathology.—In hypertrophic emphysema, idiopathic, or substantive emphysema, or the large-lunged emphysema of Jenner, the lungs are enlarged, the air cells are greatly distended with air, and the interalveolar septa are thinned and atrophic. The disease is also known by the names of chronic and diffuse emphysema.

The disease is usually encountered in persons who suffer with chronic bronchitis or whose occupations require forcible expiration with the glottis closed, as in the case of glass-blowers and players upon wind instruments. It is probable that impaired nutrition of the alveolar walls, with the result that the elastic

tissue is unable to contract and adequately expel the air from t
infundibula plays a part in the production of the disea
Freund's theory of the mechanism of production of hypertropl
emphysema assumes that it is primarily a disease of the cos
cartilages; that there is a chronic hyperplasia of these cartilag
which by ossifying prematurely cause the thoracic wall to lo
its elasticity, the emphysema of the lungs developing secondari
as a result of lack of proper support to the lungs.

Hypertrophic emphysema is a bilateral disease, and involv
both lungs to a similar degree. The distention of the lungs

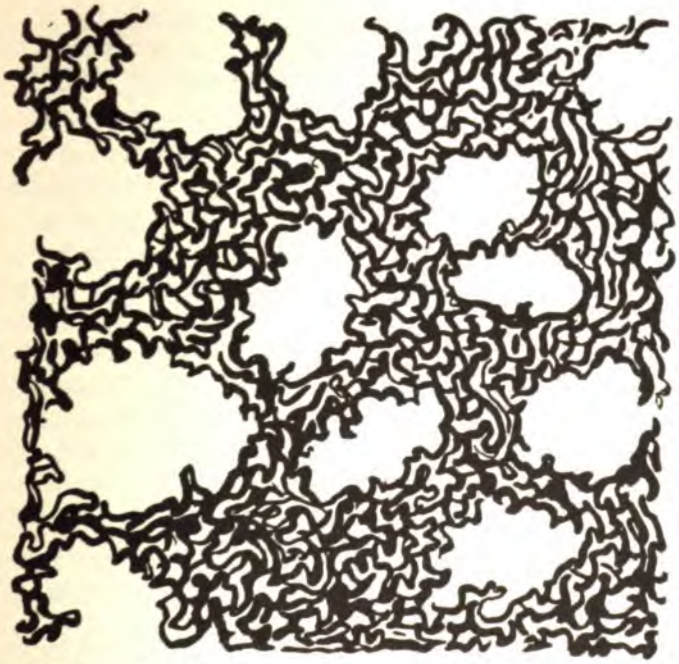


Fig. 115.—Pulmonary capillaries. The walls of the alveoli are thickly studded w
capillaries; any marked alteration of alveolar air tension will therefore have a profo
effect upon the circulation. (Brown, after Böhm, Davidoff, and Huber.)

general in all directions, but it attains its maximum degree in t
anterior pulmonary borders, which, overlapping the heart, gi
rise to a diminution of the area of that organ which is in conta
with the anterior chest and causes a consequent diminution in t
area of cardiac dullness. The lungs are enlarged; they are pa
they are light and feathery to the touch; and they do not collap
readily when the thorax is opened at autopsy.

roscopically the alveoli are observed to be abnormal large, and it is observed that in many instances the interalveolar septa are atrophic and have ruptured, leading to the production of larger cavities by the coalescence of several alveoli. With the destruction of the alveolar septa, the capillaries which they supported are destroyed, and the quantity of blood which is exposed to the air in the infundibula is commensurately diminished, resulting in deficient aeration of the blood, with consequent dyspnea and occasionally cyanosis upon moderate exertion.

The pleura covering the lung loses its pigmentation in patches, a condition which was termed by Virchow "albinism of the lung." The bronchi and bronchioles show signs of chronic bronchitis, and bronchiectatic dilatations are common.

The right heart gradually hypertrophies, as a result of the increased burden which is thrown upon it, and the tricuspid ring is usually enlarged so that the valve segments often fail to close the orifice completely.

Rupture of the atrophic walls of the infundibula subjacent to the pleura may result in the production of pneumothorax.

Physical Signs.—*Inspection.*—Hypertrophic emphysema produces a definite alteration in the size and shape of the thorax, the barrel-chest of this disease. In this type of thorax, the anteroposterior diameter is increased to such a degree that it equals or exceeds the transverse diameter. The expansion of the chest is minimal, the thorax rising and falling vertically *en masse.* The expiratory movement is of longer duration than is the inspiratory effort.

The cardiac impulse is often invisible, and there is frequently visible epigastric pulsation, while pulsation of the jugular veins is common. In the late period of the disease, when cardiac failure is imminent, the patient is dyspneic, and frequently cyanotic. Not infrequently there is a delicate tracery of distended veins over the lower portion of the thorax, produced by intrathoracic obstruction to the venous return.

The facies of hypertrophic emphysema is fairly characteristic of the disease. The eyes are slightly prominent; the nose is somewhat thickened and cyanotic; while the head is thrown slightly backward in the effort to bring the accessory muscles of respiration into play. The neck is short and thick with prominent sternomastoids and trapezii.

Litten's diaphragmatic shadow is abolished in the established case of hypertrophic emphysema; the subject presents persistent chronic cough; and the terminal phalanges of the fingers are f quently clubbed.

Palpation.—As a consequence of the excessive aerial content the lungs, vocal fremitus is diminished in intensity over both si of the thorax. However, in cases with marked chronic bronchi rhonchal fremitus is occasionally encountered. Also occasiona the intervention of the voluminous anterior pulmonary bord between the heart and the chest wall obscures the cardiac impul

Frequently there is a palpable impulse in the epigastrium wh is due to the powerfully acting right ventricle. The liver is seld displaced sufficiently to render its lower border freely palpa below the right costal arch; but in late cases, when right he failure is imminent, palpation of the liver frequently reveals systolic pulsation of tricuspid regurgitation. The spleen is rar displaced in hypertrophic emphysema. In late cases palpation the abdomen may reveal the presence of moderate ascites.

Palpation reveals the deficient expansion of the thorax; and up finger-tip palpation the intercostal spaces are hard and unyieldi

Percussion.—Percussion yields a hyperresonant note over b lungs; and the limits of pulmonary resonance are extended in directions, upward into the root of the neck, anteriorly, encroa ing upon the area of cardiac dullness, and inferiorly over the ar of hepatic and splenic dullness. Percussion is useful in detecti downward displacement of the liver, and the presence of ascites eliciting dullness in the flanks with tympany in the median line the abdomen.

The respiratory excursion of the lungs, as determined by p cussing the lower borders of the lungs during expiration and spiration, respectively, is greatly diminished in hypertrop emphysema, rarely indeed exceeding half an inch.

Auscultation.—The respiratory sounds are distant, with p longation of the expiratory phase. The expiratory murmur harsh and is not infrequently dotted with râles which are due chronic bronchitis. The inspiratory murmur is always short, a often it is entirely inaudible during quiet breathing. Vocal re nance, like vocal fremitus, is impaired over both lungs.

The heart sounds as a whole are diminished in intensity, owing the intervention of the distended anterior borders of the lu between the heart and the chest wall. Of the individual sounds, pulmonary sound is accentuated, as a result of the obstacle wh is offered to the pulmonary circulation. Late in the disease murmur of tricuspid regurgitation is often audible.

Diagnosis.—The diagnosis of hypertrophic emphysema is frequently to be made during a casual examination. It rests upon the characteristic deformity of the thorax, associated with dyspnea and cyanosis, persistent chronic cough, the short or absent inspiratory murmur, and the prolonged, harsh expiratory murmur.

Differential Diagnosis.—*Chronic bronchitis,* with its chronic cough, shortness of breath upon exertion, and slightly prolonged expiratory murmur, simulates hypertrophic emphysema; but this disease is not attended in its early stages by deformity of the thorax, neither does it as a rule show general extension of the limits of pulmonary resonance, which forms so distinctive a feature of hypertrophic emphysema. However, the two diseases frequently coexist.

Pneumothorax, which in its incipiency may resemble hypertrophic emphysema, is a unilateral affection, which develops rapidly, affording a hollow and tympanitic note upon percussion, frequently also the succussion sound, the metallic tinkle, and the coin test, which serve to differentiate it from hypertrophic emphysema.

ATROPHIC EMPHYSEMA

Clinical Pathology.—Atrophic emphysema, a pulmonary disease in which the total bulk of the lung is decreased, is a senile change, a part of the general wasting of the tissues of the body incident to advanced age. The disease, if such it may be styled, is associated with persistent chronic cough, lasting over a period of many years, and associated with chronic shortness of breath upon exertion.

In the subject of atrophic emphysema the thorax is abnormally small, the obliquity of the ribs is increased, and the excursion of the thorax during respiration is distinctly limited.

The lung as a whole is reduced in size; the pleura is deeply pigmented, and the pulmonary parenchyma shows evidence of pulmonary congestion, edema, or infarction. The bronchial tubes frequently present dilatations, which are surrounded by areas of induration.

Microscopically there is atrophy and rupture of many of the interalveolar septa, leading to the formation of larger chambers by the coalescence of several smaller ones. The capillaries which the alveolar walls supported are destroyed in the areas of alveolar rupture, decreasing the quantity of blood which is exposed to air in the infundibula.

Physical Signs.—*Inspection.*—In the subject of atrophic emp sema the thorax is small; the intercostal spaces are narrowed; ribs pursue a more oblique course than normally; the supraclavi lar and infraclavicular fossæ are of abnormal depth; and thoracic excursion during respiration is slight. The dyspnea atrophic emphysema, instead of being chiefly expiratory as hypertrophic emphysema, is mixed, the duration of the two pha of the respiratory cycle being approximately equal.

Palpation.—Upon palpation of the small thorax of the sub of atrophic emphysema vocal fremitus is as a rule moderately ex gerated, owing to the increased density of the lungs and to diminution in their air content.

Percussion.—In atrophic emphysema the limits of pulmon resonance are decreased in all directions. Even within the ar of resonance, as determined by percussion of the thorax, there moderate impairment of the normal vesicular quality of the r nance, attributable over the apices to fibrosis and condensation the pulmonary tissues, and over the bases posteriorly to ede congestion, or infarction. The area of cardiac dullness is extend as a result of shrinking of the lungs, exposing a large area of heart to the anterior chest wall. Similarly, the upper limits of areas of hepatic and splenic dullness occupy an abnormally h position, owing to contraction of the lungs.

Auscultation.—The respiratory murmur over the entire thora commonly feeble; but occasionally broncho-vesicular respiration encountered. In the presence of chronic bronchitis, which of coexists with the emphysema, the moist râles of this condition in evidence over the bases of the lungs.

Diagnosis.—The diagnosis of atrophic emphysema is rea made in an elderly subject with symmetrically diminished thor with a generally "dried up" appearance, chronic shortness breath, and chronic cough of extensive duration.

COMPENSATORY EMPHYSEMA

Clinical Pathology.—In compensatory emphysema certain p tions of a lung, or indeed an entire lung, contain an excess of as a result of a diminution of the air space of a portion of same or of the opposite lung. Usually of transient duration, c pensatory emphysema may become permanent. In inflammat

Inspection.—Upon inspection in a case in which the entire l is compensatorily emphysematous, there is unilateral bulging the thorax corresponding to the side of the emphysema; and bulging is accentuated by the retraction of the opposite side, wh is diminished as a result of disease of the lung which has occasio the emphysematous state of its fellow. Small areas of locali compensatory emphysema produce no alteration in the contour the thorax.

Palpation.—Vocal fremitus over a unilateral compensat emphysema is diminished in intensity as a result of the rarefact of the pulmonary tissues. Over the opposite lung the fremitu of variable intensity, being influenced by the state of the l which has resulted in the compensatory emphysema of its fell In the presence of consolidation the fremitus is commonly ex gerated, whereas in the presence of extensive pleural effusion is totally abolished over the effusion.

Percussion.—Percussion in the case of compensatory emphyse of an entire lung yields a hyperresonant note over the emphyse with dulling of the note over the crippled lung. Localized emp sema of limited extent produces no alteration in the normal r nance of the lung upon percussion.

Auscultation.—Over an emphysematous lung the respirat murmur is puerile, with slight prolongation of the expiratory ph Over the opposite lung the murmur is influenced by the condit which was responsible for the unilateral compensatory emphyse being bronchovesicular or frankly bronchial in the presence consolidation of the lung, and being absent over the distribution a large pleural effusion.

Diagnosis.—A diagnosis of compensatory emphysema rarely be made upon the physical signs alone, as these are quently atypical. The history of the case should be elicited, w the discovery of some adequate cause of compensatory emp sema. Hyperresonance of one side of the thorax with pue respiration and vicarious expansion, with dullness of the oppo side with possibly retraction of the thorax, and with defici expansion speaks strongly in favor of compensatory emphyse

ACUTE VESICULAR EMPHYSEMA

Clinical Pathology.—Acute vesicular emphysema is a condit in which the infundibula are acutely distended from expirat efforts or fits of coughing in the presence of an obstacle to free egress of air from the lungs. The disease is apt to develop during bronchopneumonia, bronchial asthma, tracheal and bronchial stenosis, and during the extreme dyspneic attacks of cardiac failure.

The distention of the lungs occurs abruptly; but the alveoli are merely transiently distended; and large cavities are not produced by rupture of the interalveolar septa as in hypertrophic emphysema; for recovery ensues or death occurs ere this change supervenes.

Physical Signs.—The physical signs of acute vesicular emphysema resemble in the main those of the hypertrophic form of the disease. There is general extension of the areas of pulmonary resonance; the percussion note is hyperresonant or even tympanitic; and upon auscultation there are sibilant râles universally over both lungs, with prolongation of the expiratory murmur. However, these signs are transient, and auscultation of the heart reveals no accentuation of the pulmonic second sound.

INTERSTITIAL EMPHYSEMA

Clinical Pathology.—In interstitial emphysema a variable quantity of air or gas is present in the interlobar or interlobular septa, or beneath the pleura. Air may gain access to these regions as a result of traumatism or as a consequence of violent expiratory efforts, when rupture of the lining epithelium affords ingress of air to the subjacent structures. It has also occurred during convulsions, during parturition, and as a sequence of straining at stool. It may be caused by ulceration of the bronchi, or by abscess or gangrene of the lung. Interstitial emphysema in the newly born has been caused during violent efforts to mechanically inflate the lungs, and it has resulted from spasmodic closure of the glottis.

When air or gas has gained access to the stroma of the lung, it collects in the form of beads or bullæ of variable size. In certain instances they are very small, while in other cases they attain to the size of a walnut. The beads of air are prone to make their way toward the root of the lung and into the mediastinum and thence to pass upward along the trachea and to appear beneath the skin of the base of the neck. Or the opposite sequence of events may be observed. Following tracheotomy wounds, air has entered the tissues and burrowed downward into the roots of the lungs and invaded the stroma of these organs. Not infrequently the beads of air form bullæ immediately subjacent to pleura, and by rupture produce limited pneumothorax.

Physical Signs.—Interstitial emphysema produces few phys signs, and the condition may escape detection entirely durin casual examination. When the air makes its way upward i the root of the neck and appears subcutaneously, it is apt produce a protrusion, which, upon palpation with the finger-t yields a peculiar crackling crepitus. Large beads of air subjac to the pleura are apt to yield a sound closely simulating pleural friction sound.

ABSCESS OF THE LUNG

Clinical Pathology.—The cases of pulmonary abscess wh come before the clinician may be classified as bronchogenic, pn mogenic, or extraneous in origin.

The cases of bronchogenic origin develop as the conseque of the aspiration into the bronchial tubes of infectious mate during or subsequent to operative procedures upon the buccal nasopharyngeal structures, and as the result of the lodgment the bronchi of aspirated foreign bodies.

The initial alteration in the postoperative case is the est lishment of a local bronchitis at the site of implantation of aspirated infectious material. Local ulceration of the bronc mucous membrane ensues, and is attended by thinning and yi ing of the bronchial wall, with the consequent production bronchiectasis. With the further disintegration of the bronc wall and by the extension into the bronchioles and infundibul the septic bronchitis, a deglutition or aspiration bronchopn monia is established, which is prone to eventuate in pulmon abscess.

When an aspirated foreign body becomes arrested in a br chus, local inflammation and ulceration of the bronchial muc membrane occurs at the site of lodgment, with the developm as demonstrated by Cohn, of bronchiectasis at this point, and distal to the occlusion as was formerly believed. The progress disintegration of the bronchial wall in these cases, combined w the implantation of pyogenic organisms, not infrequently is p ductive of pulmonary abscess.

In the pneumogenic group of cases are comprised the cases pulmonary abscess arising as a sequence of lobar pneumo nchopneumonia complicating influenza or other acute in tious diseases, pulmonary tuberculosis, syphilis, or actinomycosis of the lung.

Pulmonary abscess complicating lobar pneumonia occurs in two forms; namely, as a widespread purulent infiltration of the lung, and as single or multiple circumscribed abscesses. A purulent infiltration of the lung represents an extreme grade of gray hepatization with tardy resolution. Tuffier found that among forty-nine cases of pulmonary abscess treated surgically, in twenty-three lobar pneumonia only could be assigned as the cause; while, in a series of one hundred cases, Wessler found thirty-seven cases to be distinctly of pneumonic origin.

Chronic ulcerative phthisis in the course of its evolution frequently results in pulmonary abscess. The tuberculous abscess is discrete and circumscribed, develops during the latter part of the course of the disease, and is associated with caseation and cavity formation.

The cases of pulmonary abscess of extraneous origin comprise the cases of pyemic origin, and the "perforating abscesses" of Stokes. When an infectious embolus from an area of osteomyelitis or from a vegetation upon one of the cusps of a cardiac valve which is the seat of malignant endocarditis occludes a branch of the pulmonary artery, an infarct of the pulmonary tissues is apt to form and through secondary infection to eventuate in abscess of the lung. Pyemia is usually attended by the formation of multiple pulmonary abscesses of limited dimensions. They are usually situated in the periphery of the lung, immediately subjacent to the visceral pleura; and they assume primarily a conical form, with the base of the cone directed toward the pleura.

Stokes designated as "perforating abscesses" of the lung the numerous cases of pulmonary abscess in which collections of pus from extraneous sources involve the lung secondarily by contiguity of structure. The source of the pus in this class of cases is variable. It may originate in an abscess of the thoracic wall, an abscess of the liver, a perforating ulcer of the stomach, carcinoma of the esophagus, or suppuration of the mediastinal glands.

Perforating wounds of the thorax occasionally eventuate in abscess of the lung. A penetrating missile may thus carry into the lung fragments of wearing apparel or other bacteria-laden material, which results in suppuration of the pulmonary parenchyma.

In its dimensions a pulmonary abscess may be quite small or it may be very extensive, indeed, may involve the major part of a lobe of the lung. The pulmonary abscess is usually solitary; but in

pyemic cases multiple abscesses of limited dimensions are commo
dispersed widely throughout the lungs.

The shape of the pulmonary abscess is very irregular; and an
scess is occasionally divided by tissue bands into two or more ch
bers or loculi. The internal surface of the abscess is irregu
presenting mural shreds and occasionally excrescences protrud
into the cavity from a pyogenic membrane, which in turn is s
ported by a more or less well-defined fibrous capsule, which in
latter stages of the disease becomes the point of inception o
variable degree of insular sclerosis of the involved lobe of the lu

Pulmonary abscess is usually, though not invariably, situated
the peripheral portion of the lung, subjacent to the visceral pleu
and in the majority of cases it occupies the lower lobe of the lu
In the case of the postoperative abscess of bronchogenic ori
however, the abscess is not infrequently situated in the upper
middle lobe, especially in the case of children. Wessler fou
upon analysis of the distribution of the lesions in his series, t
among the postoperative aspiration cases the abscess was locali
in the upper lobe in eighteen cases, in the lower lobe in nine ca
and in the middle lobe in one instance; while among the nonasp
tion cases the abscess was situated in the upper lobe in twenty-f
cases, in the lower lobe in forty-four cases, and in the middle l
in three of the subjects.

The contents of a pulmonary abscess is purulent, contain
shreds of elastic tissue, bacteria, cholesterin crystals, and necr
debris. The organisms which are commonly present comprise
pyogenic streptococci and staphylococci, the pneumococcus, ba
lus influenzæ, and the bacillus of Friedländer, with the o
sional association of the bacillus pyocyaneus, bacillus coli c
munis, or the bacillus tuberculosis.

Spontaneous evacuation of a pulmonary abscess is apt to occur
rupture into an adjacent bronchus, in which event the purul
contents of the abscess cavity is partially expectorated, the shr
of elastic tissue in the sputum aiding materially in the diagno
or, if the abscess is situated immediately subjacent to the ple
this membrane may be penetrated, permitting the abscess to
charge into the pleural cavity with the consequent induction
pyo-pneumothorax.

When an intact pulmonary abscess occupies the peripheral
tion of the lung, closely subjacent to the visceral pleura, pleuriti
a constant accompaniment of the disease. The surface of
inflamed pleura is early clothed with fibrinous or fibrinopurul

exudate, occasionally with the subsequent supervention of frank empyema.

Bronchiectasis and pulmonary abscess are very frequently concomitant diseases. The existence of uncomplicated bronchiectasis lends a strong predisposition to the development of pulmonary abscess; while pulmonary abscess of bronchogenic origin induces a variable degree of bronchiectasis during its evolution.

Postpneumonic pulmonary abscess, whether it assumes the form of purulent infiltration or of multiple circumscribed abscess cavities, produces chronic induration of the lung with diminution in its total bulk, marked destruction of capillary circulation, and multiple bronchiectases.

Septic thrombosis of the cerebral vessels, with the induction of abscess of the brain, has proved a fatal complication in a considerable number of cases of pulmonary abscess.

Physical Signs.—The physical manifestations of pulmonary abscess vary with the character and with the distribution of the lesion. Multiple small abscesses, distributed widely throughout the lung, and likewise a very diffuse purulent infiltration, yield no distinctive physical signs by which alone the condition could surely be detected. Similarly, a solitary circumscribed abscess, which is situated in the depths of the lung and which is separated from the thoracic wall by a tolerably thick bed of crepitant pulmonary tissue, successfully eludes detection during a physical examination. It is in the cases in which the abscess is situated in the periphery of the lung, or when it is superimposed upon one of the larger bronchial tubes that a diagnosis is most readily achieved by the methods of physical examination.

Inspection.—The subject of pulmonary abscess generally presents an appearance of septic intoxication, with pallor of the skin and mucous membranes, which is not infrequently associated with a variable degree of cyanosis of the lips, ears, and finger-tips. If the abscess is at all extensive, there is acceleration of the respiration, which is shallow, and there is visible impairment of expansion of the side of the thorax corresponding to the disease of the lung.

The decubitus of the patient will vary with the site of the disease. When the abscess is situated in the lower lobe, the patient assumes the upright posture in order to maintain the sensitive mucous membrane of the communicating bronchial tubes free from the purulent contents of the abscess cavity. When, on the contrary, a solitary abscess involves the upper lobe, the subject is prone for similar reasons, to assume the recumbent or semirecumbent posture in bed.

When contact of the contents of the cavity with the bronchial mucous membrane occasions a paroxysm of cough, or when a closed abscess cavity ruptures into a bronchus, copious expectoration of fairly characteristic sputum ensues. The sputum occasionally possesses a somewhat sweetish odor, though the rule is rather for foul expectoration, which is raised in mouthfuls; but the sputum of pulmonary abscess never attains the excessive foulness of the expectoration attending pulmonary gangrene. Frequently the sputum contains shreds of elastic tissue; and, in the cases of extensive duration, crystals of cholesterin may be found.

Clubbing of the fingers is not uncommon in abscess of the lung, and in this acute disease these changes become manifest much earlier in the course of the disease than in phthisis.

Upon fluoroscopic examination, pulmonary abscess yields in the vast majority of cases a very definite shadow, which is of service in accurately localizing the disease; but in any equivocal case the examiner should be guided by the physical findings rather than by the radiogram.

Palpation.—When pulmonary abscess is situated in the peripheral portion of the lung, closely subjacent to the visceral pleura, friction fremitus which is produced by a coincident pleuritis may occasionally be detected upon palpation of the thorax. Finger-tip palpation of the intercostal spaces over this area yields a sensation of increased resistance when the abscess cavity is filled, which gives place to a sensation of markedly diminished resistance upon partial or complete evacuation of the purulent contents of the subjacent cavity.

Numerous transient variations in the intensity of vocal fremitus are encountered upon palpation of the thoracic surface over an extensive pulmonary abscess which occupies the periphery of the lung. In the cases in which the abscess cavity is closed, there is persistent diminution or abolition of the vocal vibrations in the localized area of increased resistance. When, however, the abscess possesses a patent bronchial communication by which its purulent contents is evacuated at intervals, the variations in vocal fremitus are striking. Under these circumstances tactile fremitus is absent over the filled cavity, and reappears in exaggerated form upon partial evacuation of the secretions of the abscess cavity.

Percussion.—Pulmonary abscess involving the peripheral portion of the lung yields dullness upon percussion, when it is filled, with hyperresonance over the adjacent areas as a result of compensatory emphysema of the neighboring air vesicles. When, however, the abscess has recently discharged a considerable portion of its purulent contents, the sound elicited by forcible percussion of the thorax is tympanitic or amphoric. In other words, the sounds elicited now are those of pulmonary excavation; and, if the abscess cavity possesses a patent bronchial communication, the examiner may be successful in eliciting a cracked-pot sound or the phenomena of Wintrich, Friedreich, or Gerhardt.

Auscultation.—In the presence of abscess of the lung, whether the cavity be filled or empty, auscultation of the zone surrounding the disease reveals the presence of numerous crepitant and subcrepitant râles, which arise as a consequence of a local bronchiolar inflammation and pulmonary edema. If the abscess is of considerable dimensions and is filled with secretions, the vesicular murmur is abolished over the corresponding area of the thoracic surface, while vocal resonance is markedly impaired in the same area. If, however, the abscess cavity be only partially filled, blowing bronchial or amphoric breathing is encountered, together with exaggerated vocal resonance upon auscultation.

Diagnosis.—Signs of sepsis such as chills, fever, and colliquative sweats, with copious expectoration of purulent sputum, combined with signs of pulmonary excavation, usually involving a lower lobe of the lung, following upon one of the conditions which may be provocative of pulmonary abscess, suggest the diagnosis of abscess of the lung. When a patient with lobar pneumonia continues to run a temperature with sweats after the crisis of the disease has occurred, one should think of the possible supervention of a purulent infiltration upon the pneumonic state.

Thoracic pain is frequently a prominent symptom in the cases in which the abscess is subpleural with complicating inflammation of this membrane. In cases of pyemic origin signs referable to the small, disseminated abscesses are often obscured by the general symptoms of pyemia. In the later stages of the disease emaciation is striking, with pallor of the skin and cyanosis of the lips and digits, while the patient is frequently somnolent or delirious. When, in closed cases, a bronchus is perforated and copious expectoration of purulent sputum containing elastic fibers occurs, the diagnosis is assured. Upon perforation of the visceral pleura by a subpleural abscess, signs of pyopneumothorax are in evidence.

Differential Diagnosis.—Pulmonary abscess must be differentiated from *bronchiectasis, empyema, pulmonary gangrene,* and *chronic ulcerative phthisis.*

Bronchiectasis is attended by copious expectoration of mucopurulent or purulent sputum, with frequently signs of pulmonary excavation. The sputum of bronchiectasis, however, is free from elastic shreds; and the history and course in the two affections are entirely dissimilar, bronchiectasis being a distinctly chronic disease of extensive duration and mild symptomatology, while pulmonary abscess is an acute disease of relatively brief duration and stormy course.

Empyema of extensive duration produces signs of septic intoxication with pallor, emaciation, chills, and sweats; but in empyema the disproportion between the two sides of the thorax is more extreme; there is demonstrable an extensive area of flatness laterally and posteriorly, with absence of respiratory sounds; and the sputum does not contain elastic fibers.

From *pulmonary gangrene,* which in its evolution also produces cavitation, abscess is differentiated by the absence of the extreme fetor of the expectoration of the former disease, a fetor which rapidly pervades a house or hospital ward and immediately suggests the diagnosis of gangrene. Moreover, the sputum in gangrene of the lung contains shreds of decomposing pulmonary tissue, which frequently present the characteristic structure of the alveoli of the lung.

In *chronic ulcerative phthisis* the vomicæ are prone to appear in the upper lobes, where they are encompassed by zones of infiltrated and consolidated pulmonary tissue. Phthisis is a chronic disease, and bilateral disease commonly can be detected in the presence of cavitation. In every case repeated examinations of the sputum should be made for the detection of the tubercle bacillus.

GANGRENE OF THE LUNG

Clinical Pathology.—As invasion of the lung in which the circulation is normal by pyogenic bacteria or infectious material from the upper respiratory passages is apt to eventuate in abscess of the lung, so a similar invasion of the lung in which the circulation is deficient is prone to result in pulmonary gangrene. It is upon this impairment of the vascular integrity of the pulmonary tissues that rests the predisposition to pulmonary gangrene in subjects of diabetes mellitus, chronic valvular disease of the heart, and chronic alcoholism.

Gangrene of the lung, as first recognized by Laennec, occurs in two forms; namely, as circumscribed pulmonary gangrene, and as diffuse gangrene of the lung. The two classes of cases differ as widely in their mode of inception and evolution as they do in their physical manifestations.

Circumscribed pulmonary gangrene has its point of inception in the pulmonary arteries, in the bronchi, or in cavities of bronchiectatic or tuberculous origin.

Embolic gangrene of the lung, in which infectious embolism arises as a result of malignant endocarditis, osteomyelitis, or suppurative otitis media, is of arterial origin. In this group of cases the area of gangrene is situated peripherally, not infrequently closely subjacent to the visceral pleura. Occasionally pulmonary gangrene ensues upon noninfectious embolism of the pulmonary artery.

Pulmonary gangrene of bronchial origin follows the lodgment of aspirated foreign bodies in the smaller bronchial tubes, or it ensues upon the aspiration into the finer bronchioles of infective material from the upper respiratory passages. Cohn has demonstrated that following the lodgment of a foreign body in the bronchial system, inflammation and ulceration of the mucous membrane with subsequent disintegration of the bronchial wall occurs, with the establishment of bronchiectasis, pulmonary abscess, or pulmonary gangrene as the concomitant factors may determine. Similarly, the aspiration of infective material from the upper respiratory passages is prone to induce an aspiration pneumonia or a putrid bronchitis at the area of arrestment, in which, in the words of Hensel, "the putrid properties of the aspirated particles lend a gangrenous tendency to the catarrhal pneumonia which they produce."

Gangrene of the lung arising from pulmonary excavation is encountered as a sequence of bronchiectasis with the accumulation of purulent secretions, chronic ulcerative phthisis with cavity formation, and abscess of the lung.

Traumatism in the form of penetrating wounds of the lung has caused pulmonary gangrene, as has also trauma in the form of contusion or laceration of the pulmonary tissue without perforation of the chest wall.

Diffuse gangrene of the lung is an occasional terminal complication of lobar pneumonia; but more frequently it arises as a sequence of catarrhal pneumonia of the aspiration or deglutition type. The disease has similarly been induced by carcinoma of the lung, and it has arisen as a result of pulmonary compression by aortic aneurysm and mediastinal tumor. Grisolle, in an analysis of 305 cases of lobar pneumonia, found not a single case terminating in gangrene; but he noted the great constancy of pneumonic consolidation in the area surrounding gangrenous areas of the lung, consolidation which he attributed to the proximity of the gangrenous area.

In the circumscribed form of pulmonary gangrene the disease assumes either the form of a single area of pulmonary mortification, or of multiple areas of gangrene, separated by partially crepitant pulmonary tissue. The disease most frequently involves the inferior lobe, in which it is prone to develop in the periphery of the lung. In the early stage of the disease the gangrenous area is reddish-brown, green, or black, and is firm; but in the further evolution of the morbid process softening and destruction of the pulmonary tissues occur, with the production of a central excavation with irregular, rugged walls, and which contains a greenish offensive fluid. In the subpleural cases the finger of the pathologist penetrates readily into the area of gangrenous tissue. In the cases in which the gangrene is more centrally situated, the gangrenous area is surrounded by a zone of pulmonary tissue which is the seat of local pulmonary congestion with imperfect consolidation or splenization, while adjacent to the area of congestion the lung is the seat of collateral edema.

In rapidly spreading gangrene of the lung a branch of the pulmonary artery is apt to be eroded, eventuating in pulmonary hemorrhage, which may be so copious as to prove fatal. Owing to the usual peripheral distribution of the pulmonary lesion, the visceral pleura is also apt to be perforated, with the establishment of pyopneumothorax.

Bronchitis is a constant concomitant state, as a result of the continuous irritation of the bronchial mucous membrane by the fetid contents of the gangrenous cavity. Gangrene of the lung is likewise occasionally associated with abscess of the brain. In pulmonary gangrene of bronchial origin varying degrees of bronchiectasis are frequently present in the lung, and pulmonary abscess is not infrequently complicated by gangrene of the adjacent portions of the lung.

In pulmonary gangrene there is a rapid reduction in the erythrocytes and a commensurate reduction in the hemoglobin of the circulating blood stream; and, in cases of pyemic origin, septicemia is not infrequently demonstrable. Leucocytosis is the exception and not the rule. The course of the disease is brief and stormy, the subject succumbing in the vast majority of cases in a period varying from a few weeks to a few months from the onset of his disease.

Physical Signs.—The physical signs of pulmonary gangrene vary with the site and the distribution of the lesions which are present in the lung. When the gangrenous area occupies the peripheral portion of the lung, and when there is active pulmonary excavation, the physical signs are those of cavity, the manifestations of the disease varying naturally as the cavity possesses a patent bronchial communication or is closed. When, on the contrary, the area of gangrene is centrally situated, is devoid of all communication with the bronchial system, and is separated from the thoracic parietes by a tolerably thick bed of crepitant pulmonary tissue, the physical signs are anomalous or entirely wanting. In the majority of cases, in addition to the signs which are attributable to the gangrenous process, the characteristic signs of coexistent bronchial inflammation and partial consolidation are in evidence.

Inspection.—The subject of pulmonary gangrene commonly presents a picture of extreme septic intoxication. Emaciation is rapid and the skin is pallid, frequently with cyanosis of the lips, auricles, and digits. The patient is wracked by persistent paroxysms of cough, arising as a result of irritation of the sensitive bronchial mucous membrane by the acrid contents of the gangrenous cavity.

The patient instinctively assumes the posture in which the contents of the cavity is prevented from coming in contact with the mucosa of the communicating bronchi. When the gangrene attacks an inferior lobe of the lung, the patient is prone to assume the semirecumbent posture; whereas, when the area of gangrene is situated in an upper lobe, the subject rests most comfortably in the recumbent posture with the shoulders lower than the remainder of the trunk.

Hemoptysis is frequently to be noted in gangrene of the lung. It may consist in an admixture of traces of blood with the sputum, or it may occur in the form of copious pulmonary hemorrhages.

The breath is exceedingly foul in pulmonary gangrene, a fact which constitutes a valuable sign of the disease when considered in conjunction with the sputum, which is characteristic of the condition. The latter is abundant; and, upon standing for several hours, separates into three strata: an inferior stratum composed of heavy or brown sediment containing shreds of elastic tissue, bacteria, blood cells, fatty-acid crystals, leucin and tyrosin; a median layer composed of brown or greenish serous fluid; and a superior stratum of grayish-brown froth.

Fluoroscopy reveals the distribution and dimension of the gangrenous involvement of the lung.

Palpation.—In the subject of pulmonary gangrene the inspiratory excursion of the thoracic wall is strikingly diminished; the respiratory movements are at the same time more frequent and often irregular or jerking; and the lifting power of the thoracic cage is practically nil.

When, in the presence of central gangrene of the lung, the adjacent zone of infiltration and consolidation attains to the visceral pleura, there is frequently appreciable to the palpating hand a minor exaggeration of vocal fremitus, an increase which is further exaggerated when adhesions have been established between the visceral and the costal pleura. When pulmonary gangrene originates in the cavities of chronic ulcerative phthisis, bronchiectasis, or pulmonary abscess, occupying the peripheral portion of the lung as these lesions are prone to do, tactile fremitus is markedly exaggerated over the site of the lesion, and to a lesser degree over the adjacent zone of consolidated pulmonary tissue.

Percussion.—The percussion findings in cases of pulmonary gangrene have been variable; and, unless they are construed in connection with other manifestations of the disease, they are apt to result in confusion, especially in cases of closed or latent gangrene of the lung. When the gangrenous area is covered by a bed of densely consolidated pulmonary tissue, the examiner encounters dullness upon percussion, a dull note upon which there is not infrequently engrafted a tympanitic quality when the gangrene has resulted in pulmonary excavation. A suggestive feature of the dullness which attends pulmonary gangrene resides in the fact that the area of impaired resonance frequently extends uninterruptedly over an interlobar fissure, as gangrene is frequently not limited to a single pulmonary lobe, but is prone to involve contiguous portions of adjacent lobes of the lung.

Careful percussion of the thoracic surface will frequently reveal a zone of moderate hyperresonance surrounding the area of dullness which is induced by the pulmonary consolidation, a hyperresonance which is to be attributed to local compensatory emphysema of the pulmonary alveoli in these regions.

Auscultation.—The vesicular murmur in pulmonary gangrene is frequently distant and feeble over the entire thorax as a result of the general debility of the subject of the disease. Occasionally bronchial breathing is to be elicited over an area of consolidation or pulmonary excavation. The crepitant râle is very frequently in evidence as an indication of the concomitant bronchial inflammation.

In the presence of gangrene of the peripheral portion of the lung a fine pleural friction sound is occasionally to be detected, a rub which develops as a consequence of a complicating local pleuritis.

In cases of embolic origin auscultation of the precordia not infrequently reveals the murmurs of coexistent valvular lesions.

Diagnosis.—The signs and symptoms upon which the diagnosis of pulmonary gangrene must be based comprise progressive emaciation and prostration, dyspnea, elevation of temperature, rapid cardiac action, foulness of the breath, the suggestive decubitus of the patient, and the characteristic sputum, when it is present. The occurrence of hemoptysis and the detection of shreds of disintegrated alveolar tissue in the expectoration are determining factors in arriving at a diagnosis. But in cases of latent pulmonary gangrene the breath is not foul, neither is the sputum characteristic, as in this class of cases the area of gangrene is definitely circumscribed and is devoid of any communication with the bronchial system. This type of the disease is prone to develop in diabetic and insane patients, and in these cases an error in diagnosis is almost certain to arise. A history of diabetes, bronchopneumonia, valvular heart disease, or bronchiectasis should serve in some measure to place the physician upon his guard when interpreting obscure physical findings in a debilitated subject.

Differential Diagnosis.—Mere foulness of the breath in any suspected case of pulmonary gangrene does not suffice to establish a positive diagnosis, as a foul breath may result from putrid bronchitis, decomposition of the purulent contents of bronchiectatic cavities, or even from carious teeth, in the absence of gangrene of the lung.

In *pulmonary abscess,* while the sputum is abundant and purulent, the odor of the breath is sweetish rather than fetid, as in gangrene. Moreover, the sputum in abscess is copious, occurring frequently as "mouthful expectoration;" it more frequently contains bacteria and cholesterin crystals; and shreds of alveolar tissue are not as abundant as in the case of gangrene.

Chronic ulcerative phthisis with cavity formation is attended by foulness of the breath and purulent expectoration containin elastic fibers; but the sputum in this disease contains tubercl bacilli, and the foulness of the breath never approaches the stenc of pulmonary gangrene. The vomicæ of phthisis are prone t involve the upper lobe, and at this advanced stage of the diseas bilateral involvement of the lungs is demonstrable. Unfortunately in gangrene of the lung acid-fast bacilli closely resembling th tubercle bacillus morphologically are apt to be encountered in th sputum and to result in an erroneous diagnosis.

In *putrid bronchitis* the breath is excessively foul; but in thi disease the expectoration which is raised is free from elastic shreds while it contains characteristic elements in the form of Dittrich' plugs. While the prostration attending certain cases of putri bronchitis is remarkable, it does not equal that of advanced gangrene of the lung.

TUMORS OF THE LUNG

Clinical Pathology.—Tumors of the lung may arise primaril in this organ, or may be secondary to tumor arising elsewhere i the body and implicating the lung as a result of metastasis. O the two varieties of pulmonary neoplasms, the primary form i decidedly rare, and the secondary is the usual type of tumor o the lung.

The *primary tumors* of the lung comprise carcinoma, sarcoma and endothelioma. Carcinoma in its evolution involves usuall one lung, where it forms a large mass, and later breaks down wit the formation of a pulmonary cavity. But in other instances ther develops instead a diffuse cancerous infiltration of the lung, simulating in its physical manifestations chronic ulcerative phthisis

The *secondary tumors* of the lung comprise all varieties of malignant growths. Secondary carcinoma of the lung rarely forms large single tumor, but is usually multiple, and not uncommonl involves the pleura. The carcinomatous nodules are diffusel scattered over both lungs. This disease represents metastases fro a primary tumor which may be situated in the breast, the gastrointestinal tract, the genitourinary tract, or bone. Hodgkin's diseas may affect the lung, traveling by way of the mediastinal and bronchial lymphatic glands.

Carcinoma of the lung produces swelling of the bronchial an mediastinal glands and occasionally of the glands of the neck Pleurisy is a common complication or accompaniment of pulmonar

CHAPTER XI

DISEASES OF THE PLEURA

ACUTE FIBRINOUS PLEURISY (ACUTE PLASTIC PLEURISY; PLEURITIS SICCA)

Clinical Pathology.—Acute fibrinous pleurisy, acute plastic pleurisy, or pleuritis sicca, occurs in a primary form and as a secondary disease.

Primary acute fibrinous pleurisy is frequently noted following exposure to cold, particularly in patients who are debilitated by the excessive use of alcohol, or from other causes. Primary pleurisy has also followed violent contusion of the thorax.

Secondary acute fibrinous pleurisy occurs secondarily to disease of the lung, notably lobar and lobular pneumonia, and secondarily to disease in more remote portions of the body. It develops in connection with tuberculosis of the lungs or of the bronchial glands, bronchiectasis, pulmonary infarction, and in abscess and gangrene of the lung. Acute fibrinous pleurisy also occurs as a complication of the acute exanthematous fevers and occasionally during the course of other acute infectious diseases.

Among primary lesions without the lungs which may be followed by acute fibrinous pleurisy may be mentioned endocarditis, pericarditis, tonsillitis, pyorrhea alveolaris, arthritis, and typhoid fever.

The inflammation usually involves the lower lateral and anterior portions of the pleura, in which site it may be localized to a very limited area, or it may involve the greater portion of the visceral pleura. Under the influence of the inflammatory process the pleura becomes dull and lusterless, with a rather granular, irregular surface. The membrane is thickened and the surface is covered with one or more layers of fibrinous exudate. During the movements of respiration the exudate is apt to be rolled into folds upon the surface of the pleura, or it may be thrown up into exuberant masses. There is a small amount of cloudy fluid exuding from the inflamed surface; but it never attains the degree which is seen in serofibrinous pleurisy or pleurisy with effusion.

Microscopically, the pleura presents desquamation and degeneration of the covering endothelium at the site of the inflammation, patches of the pleura being found entirely devoid of endothelial covering. At the same time, the subserous connective tissue layer of the membrane is edematous and exhibits a variable degree of leucocytic infiltration. The vessels in the zone of the inflammation are dilated and are filled with erythrocytes. Upon microscopic examination of the exudate which is thrown out, it is found to contain fibrin, serum, and a variable number of pus cells.

In cases of acute fibrinous pleurisy of extensive duration the visceral and parietal layers of the pleura not infrequently become adherent, impairing the movement of the lung to a variable extent during respiration.

Physical Signs.—*Inspection.*—In acute fibrinous pleurisy the respirations are moderately accelerated and are apt to be jerky and irregular. The thoracic expansion upon the side of the disease is limited, and the diaphragmatic shadow is abolished. The trunk is commonly inclined toward the side of the disease, with slight drooping of the corresponding shoulder.

Palpation.—Palpation of the thorax reveals in many instances pleural friction fremitus, which is produced by the rubbing together of the roughened surfaces of the visceral and parietal pleuræ. If the pleural inflammation involves the portion of the visceral pleura which overlies the pericardium, the friction fremitus assumes the pleuropericardial type. Vocal fremitus seldom presents any alteration in acute plastic pleurisy; but, if there is considerable pleural thickening, the intensity of the vocal vibrations is diminished over the area of the disease. Palpation confirms the presence of minor deficiencies of expansion of the affected side of the thorax.

Percussion.—The note which is elicited upon percussion of the thorax over an area of acute fibrinous pleurisy in an initial attack of the disease betrays no departure from normal pulmonary resonance; but, in the case of repeated attacks, there may be sufficient pleural thickening to cause moderate impairment of resonance upon the side of the disease. Percussion of the inferior pulmonary borders during inspiration yields a pertinent sign of the disease in the limitation of the respiratory excursion of the diseased lung. Pain is not infrequently elicited upon percussion in this disease.

Auscultation.—Auscultation over the site of the disease reveals the pathognomonic sign of the disease, the pleural friction sound; and, in suitably placed lesions, pleuropericardial friction. The vesicular murmur is retained, but its intensity is diminished upon the side of the disease as a result of the pain which attends full inspiration. In the presence of considerable pleural thickening the murmur is abolished over the site of the thickened pleura. Vocal resonance is as a rule unchanged; but in the presence of excessive pleural thickening, its intensity is diminished.

Diagnosis.—The pleural friction sound, when it is elicited, is pathognomonic of acute fibrinous pleurisy. The disease is also attended by moderate fever and by darting, stabbing pain, which is most commonly referred to the region of the axilla or the nipple, and which is accentuated by coughing and by deep inspirations, signs which are not pathognomonic of the disease.

Differential Diagnosis.—*Pleurodynia* is attended by pain in the side, but the pain of this disease is continuous and is aggravated by movement of the trunk as well as by the movements of respiration. Moreover, there is absence of the friction sound and the localization of the pain is not as distinct and clear-cut as it is in pleurisy, the pain occasionally ceasing upon one side of the thorax to appear upon the opposite side of the chest. Pleurodynia is not attended by febrile movement.

Intercostal neuralgia is characterized by sharp, paroxysmal pain over the distribution of the nerve trunks. In this condition there are points of tenderness upon palpation over the points of exit of the nerves upon the lateral and anterior chest walls. Developing as the disease does most frequently upon a neurotic basis, pleural friction and fever are absent in intercostal neuralgia.

SEROFIBRINOUS PLEURISY (PLEURISY WITH EFFUSION; PLEURITIS EXUDATIVA)

Clinical Pathology.—Serofibrinous pleurisy, pleurisy with effusion, or pleuritis exudativa, frequently follows exposure to cold or a severe wetting, when it is attributed to an attack upon the pleura by organisms which are present in the bodily economy; but in certain instances exposure is the only apparent cause of the disease.

Lobar pneumonia, by involving the pleura over a consolidated lobe, often causes serofibrinous pleurisy; but there is occasionally seen a primary pleurisy with effusion which is caused by the pneumococcus and which arises independently of disease of the lung. Serofibrinous pleurisy is an occasional complication of nephritis and acute rheumatic fever, in which it is due either to the associated toxemia or to the bacteria which are associated with these diseases.

The organism which is most frequently associated with serofibrinous pleurisy is the tubercle bacillus. In these cases the tuberculous focus may be situated in the lung or in a distant portion of the body. The streptococcus pyogenes is the causative agent in certain cases of serofibrinous pleurisy, with or without the coincident development of a streptococcic bronchopneumonia. In the female subject pleurisy with effusion has developed in subjects with tuberculous salpingitis.

Pleurisy with effusion attacks males with greater frequency than females, usually attacking persons between twenty and fifty years of age, though no age is exempt from the disease.

In serofibrinous pleurisy there is an initial dulling and loss of luster with roughening of the surface of the pleural membrane; and this is succeeded in a few hours by the exudation of a serofibrinous exudate. In a period varying from a few hours to several days there is more or less copious exudation of serous fluid from the surface of the inflamed pleura.

The fluid gravitates to the dependent portions of the pleural sac, and mounts up higher and higher as the effusion develops. In certain instances the effusion becomes so excessive that it reaches the level of the clavicle. The lung, which is compressed by the increasing fluid, is crowded into the upper and posterior portions of the pleural cavity, occupying a comparatively small area in the upper portion of the cavity near the vertebral column.

The effusion consists of straw-colored fluid possessing a specific gravity of approximately 1,020, containing flocculi of fibrin together with numerous desquamated endothelial cells, pus cells, bacteria and blood cells. Upon withdrawal of the fluid, spontaneous coagulation is occasionally noted. The solid constituents of the effusion occupy the dependent portion of the pleural sac, and the fibrinous material adheres to the surface of the pleura, in which position when the fluid is absorbed or is withdrawn, it aids in the formation of fibrous adhesions between the visceral and the parietal pleura.

These adhesions vary in distribution in individual cases. In certain instances they are relatively few and are local; in other cases they are universally distributed over the entire pleura except for a pocket here and there; while in yet other instances they may obliterate the pleural sac entirely, resulting in chronic adhesive pleurisy. When a patient with numerous adhesions has a second attack of serofibrinous pleurisy, only the nonadherent portions of the membranes are involved, with the formation of loculated, sacculated, or encysted pleurisy.

In serofibrinous pleurisy the amount of the effusion vari from a few ounces to four liters. After pursuing a variabl course, it tends to spontaneous absorption, often leaving exte sive pleural adhesions in its wake.

In cases of serofibrinous pleurisy which are associated wit excessive effusion, there is visceral displacement. The liver the spleen is displaced downward and the heart is displace toward the side opposite the effusion.

Physical Signs.—*Inspection.*—In serofibrinous pleurisy the re piratory excursion upon the side of the effusion is restricted or abolished. With all large effusions there is unilateral bulging the chest wall and the intercostal spaces are obliterated upon th side of the disease. Litten's diaphragmatic shadow is absent.

In effusions of the right pleural sac the cardiac impulse displaced toward the left, and is occasionally elevated to th fourth interspace. It is not infrequently localized in the le midclavicular line, or even in the left axillary region. In th case of left-sided effusion, on the other hand, the cardiac impul is displaced toward the right, and it often occupies a positio behind the sternum. In extreme cases, however, the apex bea may be visible to the right of this bone in the third or fourt intercostal space.

The respirations are shallow and accelerated, as a result compression of the lung by the effusion, with consequent dimin tion in the air space. There is visible scoliosis, the vertebr column deviating toward the side of the effusion. The soun side of the thorax expands vicariously during inspiration as result of compensatory emphysema.

The decubitus of the patient is not infrequently suggestive i serofibrinous pleurisy. During the incipient stage of the diseas prior to the pouring out of the effusion, the patient is apt to l upon the sound side in the effort to protect the sensitive pleur from pressure, while, after the effusion is established, he usuall lies upon the side of the effusion in order to facilitate the fu expansion of the sound lung. The shoulder upon the side of th effusion is upon a slightly higher level than is its fellow. Sim larly, the nipple and the scapula upon the side of the effusio are farther from the median line than on the opposite side of th thorax.

Palpation.—During the incipient stage of the disease, prior the development of the effusion, palpation of the thorax reveals the presence in most cases of pleural friction fremitus. If, indeed, the lappet of lung which overlies the heart is involved, there is pleuropericardial friction fremitus. Friction fremitus is demonstrable during the incipient, dry stage of the disease; it usually disappears with the development of the effusion; and it reappears with the absorption or after the withdrawal of the fluid. Yet not infrequently friction fremitus can be detected during the height of the effusion along its upper level, where the inflamed pleural membranes come in contact. The friction fremitus not infrequently persists for years after recovery from the disease, as many subjects of the disease can attest.

Vocal fremitus varies according to the degree of the associated effusion. In the presence of moderate effusion, which does not fill the pleural sac, vocal fremitus is normal above the level of the effusion, while over the area corresponding to the effusion it is abolished. Posteriorly, near the vertebral column, over the area which is occupied by the compressed lung, vocal fremitus at the height of the effusion is apt to show exaggeration. In interpreting the intensity of vocal fremitus in this disease it must be borne in mind that dense pleural adhesions traversing a pleural effusion will and do transmit the vibrations to the palpating hand despite the presence of fluid in the pleural sac.

Palpation of the precordia confirms displacement of the apex-beat, and palpation of the lateral thoracic regions shows deficient expansion of the diseased side. There is seldom edema of the chest wall in serofibrinous effusion, this sign being more commonly present in purulent effusions. In effusion of the right pleural sac palpation reveals the inferior border of the liver at an abnormally low level below the right costal margin.

Percussion.—During the incipient stage of serofibrinous pleurisy the percussion note is unchanged. In the further evolution of the disease, however, as the effusion develops, there is a progressive impairment of vesicular resonance, finally amounting to flatness over the effusion. Percussion of the thorax immediately above the level of the effusion elicits Skodaic resonance.

With the patient in the upright posture, Ellis' line of flatness, indicating the upper limit of the effusion, can occasionally be mapped out by percussion. In pleurisy with effusion this line, which represents the upper limit of the effusion is not horizontal; it is higher posteriorly than anteriorly; and in effusions of moderate degree the line begins low down in the posterior region of the thorax and proceeds upward and forward in a curve resemblin the letter "S" to the axillary region, and thence proceeds in gradual decline to the sternum.

Grocco's triangle of paravertebral dullness is demonstrable i most cases of serofibrinous pleurisy. This triangular area, with width of two to five centimeters, with its apex directed upwar occupies the side of the thorax opposite the effusion at the level the 11th and 12th ribs. It is probably due to displacement of tl mediastinal structures by the pressure of the effusion. (See Fi 53, p. 128.)

In right-sided effusion the dullness of the fluid blends ant riorly and laterally with the dullness of the liver; whereas a effusion of the left side encroaches upon the tympany of Traube semilunar space. In serofibrinous pleurisy it is only occasional possible to detect movable dullness upon change of posture. Whi not always present in this disease, movable dullness when elicit is an infallible sign of fluid in the pleural cavity.

Forcible percussion over the upper portion of the lung, abo the level of the effusion, occasionally elicits a cracked-pot soun produced by the sudden forcible expulsion of air from the r laxed lung. Similarly, upon strong percussion over the infr clavicular region in the presence of large effusions, William tracheal tone may sometimes be elicited.

During absorption of the effusion the dullness gradually superseded by normal vesicular resonance, save at the bas posteriorly, where the resonance is apt to remain impaired for long period. Areas of impaired resonance elsewhere point areas of pleural thickening or to encysted fluid.

Auscultation.—In the incipient stage of the disease, prior to tl development of effusion, a pleural friction sound is frequent audible upon auscultation. It is usually best detected in the low axillary or mammary region. The friction sound usually thou not invariably disappears at the height of the effusion, to becon once more audible with the inception of absorption of the effusio When the portion of the pleura which overlies the pericardium involved, a pleuropericardial friction sound is audible.

The vesicular murmur is abolished over the area of the thor which overlies the effusion, while above the effusion, the respirato sounds are puerile. While this statement as a rule holds true, y in very large effusions there is occasionally distant bronchi breathing over the effusion, due to the dense compression of tl lung by the fluid which occupies the pleural cavity. The res ratory murmur over the sound lung is exaggerated as a result of compensatory emphysema.

Vocal resonance over the effusion is abolished, unless a patch of pleura be bound to the chest wall by adhesions, in which event the resonance is of approximately normal intensity in the area in question. Upon auscultation just above the level of the effusion during phonation, particularly near the angle of the scapula posteriorly, egophony is frequently elicited. Baccelli's sign, the transmission of the whispered voice through a serous while not through a purulent effusion serves to differentiate the latter condition from the former.

Upon auscultation of the heart, the sounds are often rather diffusely audible, owing to cardiac displacement. The pulmonic sound is usually accentuated and a systolic murmur may be audible, which is produced by traction upon the vessels by the cardiac displacement.

Mensuration.—Mensuration of the thorax reveals an increase in the circumference of the diseased side of from one-half to one and one-half inches. In interpreting the findings of mensuration allowance must be made for the fact that the right half of the thorax is normally larger than is the left side.

Diagnosis.—The diagnosis of serofibrinous pleurisy rests upon the deficient expansion and frequently the unilateral bulging of the diseased side, the presence of the initial friction sound which disappears with the advent of effusion, the absence of vocal fremitus over the effusion, the flat percussion note over the fluid, the absence of respiratory sounds over the effusion, with the presence of puerile or bronchial sounds elicited above the level of the fluid, and the presence of visceral displacement, together with certain special phenomena such as bronchophony, egophony, or Baccelli's sign. The presence of fluid in the pleural cavity and its character are determined by exploratory puncture. But exploratory puncture does not in every positive case reveal the presence of fluid. Even if the needle is inserted in an area of flatness, it may penetrate a region where a thickened pleura is adherent to the chest wall and so fail to secure fluid, though fluid is present.

Differential Diagnosis.—Serofibrinous is occasionally differentiated from lobar pneumonia with difficulty. The points of differentiation between these diseases have been enumerated under lobar pneumonia.

From large *pericardial effusion*, serofibrinous pleurisy is sometimes differentiated with difficulty, particularly in the cases of effusion into the left pleural cavity. But in pericardial effusion the base of the lung yields resonance instead of flatness; there is Skodaic resonance over the adjacent portion of the lung which is compressed by the effusion; the cardiac impulse is not displaced to the right; the heart sounds are feeble; the pulse is apt to be of the pulsus paradoxus type, trailing off toward full inspiration; and the degree of dyspnea is extreme and out of proportion to the extent of the effusion. Moreover, in pericardial effusion the area of dullness occupies the precordia and is pear-shaped with the base resting upon the diaphragm.

Unilateral hydrothorax presents physical signs which are largely identical with those of serofibrinous pleurisy. But in hydrothorax there is absence of the initial friction sound; there is no primary stitch in the side, or fever, but instead there is a history of heart disease, or of nephritis. Moreover, hydrothorax is frequently attended by edema of dependent portions of the body.

Ellis' curve is not present in hydrothorax and upon exploratory puncture the fluid of hydrothorax is more serous, and is of lower specific gravity; it contains less than three per cent of albumin; and it never coagulates spontaneously upon standing. In hydrothorax movable dullness is usually readily elicited, as the fluid of hydrothorax readily shifts with change of posture, while in pleurisy with effusion this sign is elicited with difficulty if, indeed, it is elicited at all.

Intrathoracic neoplasms may simulate serofibrinous pleurisy. They frequently produce displacement of the cardiac impulse, as does pleurisy with effusion. The dullness of neoplasm, however, occupies the upper portion of the thorax and is of minor extent, and it is surrounded by a zone of compressed lung, yielding Skodaic resonance. Vocal fremitus and vocal resonance are increased rather than diminished. The breath sounds are frequently suppressed, and at other times distinctly bronchial. Pulmonary neoplasm often coexists with moderate pleural effusion. Malignant neoplasms are prone to produce glandular enlargement in the supraclavicular region, and are eventually attended by emaciation and cachexia. These growths also cause enlargement of the mediastinal glands, producing in this wise pressure paralysis of the recurrent laryngeal nerves. Moreover, in the case of neoplasm, the physical signs are not influenced by change of posture.

Hepatic enlargement from abscess, echinococcus cyst, or enlargement from subphrenic abscess may simulate pleurisy with effusion. But the upper limit of dullness is immovable; it presents a convex outline directed upward; and it frequently exhibits a friction sound in the midst of the area of dullness, which would not be true if the pleural surfaces were separated by effusion. These conditions are not infrequently attended by moderate pleural effusion and hence coexistent therewith. Upon exploratory puncture the pus from an hepatic abscess may perhaps contain liver cells or bile, and occasionally amebæ. Grocco's sign is absent in hepatic enlargement. Occasionally hydatid fremitus may be elicited upon percussion over an echinococcus cyst of the liver.

Pneumothorax produces unilateral bulging of the thorax with immobilization; but the percussion note is hyperresonant or tympanitic; and the disease is attended by characteristic physical signs, as the coin test, the metallic tinkle, and the succussion sound.

LOCAL PLEURISY

Under the head of local or circumscribed pleurisy are embraced *diaphragmatic pleurisy; loculated, sacculated, or encysted pleurisy; and interlobar pleurisy.*

DIAPHRAGMATIC PLEURISY

Clinical Pathology.—In diaphragmatic pleurisy the inflammation is limited to the parietal pleura which invests the superior surface of the diaphragm and to the visceral pleura which is in contact with it.

The pleural inflammation as a rule assumes the dry, plastic type; but there is sometimes moderate effusion, which may be serous or purulent in character.

Physical Signs.—The physical signs of diaphragmatic pleurisy are slight in comparison with the subjective symptoms, which are unusually severe in their manifestations. There is urgent dyspnea; and the lower region of the thorax is fixed, moving very slightly with respiration. A friction sound can occasionally be elicited over the hepatic area in right pleural inflammation, or over Traube's semilunar space in left-sided inflammation.

There is tenderness upon pressure upon the lower intercostal spaces near the vertebral column, and extreme pain upon pressure over the insertion of the diaphragm at the tenth rib. There is occasionally tenderness upon pressure over the course of the phrenic nerve in the cervical region. Dysphagia is occasionally present, as a result of involvement of the esophageal orifice in the diaphragm; and hiccough and vomiting may accompany left-sided diaphragmatic pleurisy. The vomiting and respiratory movements exaggerate the pain of the disease. Pain is constant in the epigastric region, simulating in this respect acute disease of the abdominal viscera. The diaphragm is fixed, and the respiration is purely costal in type.

Diagnosis.—The diagnosis of diaphragmatic pleurisy rests upon the great severity of the subjective symptoms and the meagerness of physical signs. Gueneau de Mussy states that pain extending from the tenth rib to the ensiform cartilage is pathognomonic of diaphragmatic pleurisy. Andral has noted cases attended by excessive dyspnea and attacks simulating angina pectoris.

LOCULATED, SACCULATED, OR ENCYSTED PLEURISY

Clinical Pathology.—This type of pleurisy may be serofibrinous, but is more frequently purulent. In this form of pleurisy the fluid is circumscribed by adhesions between the visceral and parietal pleuræ into one or more pockets or loculi, which may or may not communicate with one another. While these loculi of effusion may develop in any portion of the pleural cavity, they are most frequently situated in the region between the midaxillary line and the midspinal line, or upon the thoracic aspect of the diaphragm. In these cases the fluid may be bounded by adhesions, the result of a previous attack of pleurisy; or an empyema may become limited and circumscribed by newly formed inflammatory adhesions.

Physical Signs.—The physical signs in this type of pleurisy are few and are frequently atypical. Areas of dullness may be elicited in certain cases; but this is by no means the rule; and vocal fremitus may be clearly transmitted by the adhesions despite the presence of fluid in the pleural cavity. Fluoroscopy and the free use of the aspirating needle are the surest means of diagnosis.

INTERLOBAR PLEURISY

Clinical Pathology.—In the evolution of serofibrinous or purulent pleurisy the pleura clothing the interlobar fissures of the lung is also implicated in the inflammation, and not infrequently become adherent, enclosing between the two pleural layers a variable quantity of serofibrinous or purulent fluid.

Also in cases of lobar pneumonia and pulmonary tuberculosis an interlobar pleurisy may occur, with adhesions of the pleural surfaces and retention of effusion. Interlobar pleurisy is usually purulent, and in its physical manifestations it frequently closely simulates pulmonary abscess. Such a collection of pus may perforate and discharge into a bronchus and lead to the expectoration of purulent sputum.

Interlobar pleurisy develops with the greatest frequency near the root of the right lung, involving the pleura which dips into the fissure between the upper and middle lobes of the lung.

Physical Signs.—The physical signs of interlobar pleurisy are frequently atypical and confusing. As a rule, there is little or no dulness upon percussion; but in certain cases a zone of dulness corresponding to the course of the fissure between the upper and middle lobes of the right lung can be found, limited above and below by a zone of Skodaic resonance.

Diagnosis.—The x-ray is of aid in the diagnosis. Aspiration is dangerous, as the lung is apt to be infected during withdrawal of the needle. The clinical picture frequently closely simulates that of pulmonary abscess.

PURULENT PLEURISY (EMPYEMA)

Clinical Pathology.—Purulent pleurisy or empyema occurs in rare instances as a primary disease, chiefly in young infants; but in the vast majority of cases it is secondary to disease or injury of the lung or thorax.

Purulent pleurisy complicates many acute infectious diseases, as scarlatina, lobar or lobular pneumonia, and pulmonary tuberculosis, and it may be a sequela of abscess or gangrene of the lung. It may be caused by penetration of the chest wall by a fractured rib or by other perforating wound. Perforation of the diaphragm by subphrenic abscess is frequently followed by empyema; and carcinoma of the esophagus is prone to penetrate the pleura and may be productive of empyema.

While it is quite possible by secondary infection for a primarily serous pleural effusion to become purulent, yet empyema is usually purulent from the outset of the disease. The pneumococcus is responsible for the greater number of cases of empyema, either having its inception as lobar pneumonia, or attacking the pleura primarily. Next in the order of their frequency come the pyogenic micrococci, the tubercle bacillus, the bacillus influenzæ and the colon bacillus. Empyema is especially frequent in young children, although no age is exempt from the disease.

In purulent pleurisy the pleural cavity contains a variable amount of pus, which is frequently quite large, often amounting to two or more liters. The solid constituents of the purulent effusion gravitate toward the dependent portions of the pleural sac, while the upper strata consist of turbid fluid. This fact may be productive of an error in diagnosis; because, if the aspirating needle is entered above the level of the distinctly purulent stratum, only turbid fluid may be withdrawn, suggesting the presence of a serofibrinous effusion. While the separation of the fluid into two strata obtains during the early stage of the effusion, yet in cases of long standing the fluid is uniformly thick and purulent and contains shreds of fibrin. Depending upon the character of the infecting organism the pus may be odorless or may be extremely fetid.

In the evolution of the disease, subsequent to the development of the purulent effusion, the tendency is toward spontaneous absorption; and it is possible for the effusion to be absorbed in its entirety. Following absorption of the effusion the pleura undergoes thickening from organization of the fibrinous layer with which it is clothed, tending to impair the transmission of sounds arising within the lung. The pleural thickening is most pronounced in the visceral layer of this membrane.

Pleural adhesions are almost invariably present in empyema, which may represent the remnants of a former pleurisy, or which may be of recent formation, serving to circumscribe the effusion into one or more pockets or loculi. They are situated principally over the upper portions of the lung, probably because this portion of the pleura is in contact, while the lower portion is separated by the effusion. The adhesions may be limited, or they may be numerous, extending well down toward the base of the lung.

Purulent pleurisy is constantly attended by changes in the lung. The effusion occupies space in the pleural cavity which was previously occupied by the lung, so that this organ is compressed, and its expansion is materially restricted. This tends toward condensation and atelectasis of the lung, and in large effusions the lung occupies a small portion of the pleural cavity near the vertebral column. In the further evolution of the disease the lung becomes splenized, and finally cirrhotic, airless, and of dark color.

The heart is displaced by the effusion, and the impact of this organ against the fluid during ventricular systole causes "pulsating empyema." The liver and spleen are apt to be displaced downward.

Extensive purulent pleurisy produces permanent deformity of the thorax. As the effusion accumulates, the side of the thorax corresponding to the disease bulges, and the intercostal spaces are obliterated or actually bulge from the pressure which is exerted upon the intercostal muscles. The diaphragm is depressed and its excursion is limited, resulting in an increase in the vertical diameter of the thorax. Following the absorption or evacuation of the purulent effusion, the intercostal spaces are retracted, and the side of the thorax corresponding to the disease partially collapses, the shoulder droops, and the spinal column is bowed toward the side of the disease.

The pus of empyema is apt to burrow beneath the costal pleura and to point subcutaneously, constituting "empyema necessitatis." Spontaneous evacuation of the effusion is apt to occur, unless it is relieved by prompt aspiration. Or, on the other hand, the pus may perforate the visceral pleura and discharge into a bronchus, with the production of pyopneumothorax.

Physical Signs.—*Inspection.*—The physical signs of purulent pleurisy which are noted upon inspection are in the main very similar to those of serofibrinous pleurisy; but the diseased side presents a greater degree of unilateral bulging, and there is more apt to be bulging of the intercostal spaces, particularly over the lower regions of the thorax. Edema of the chest wall is noted when the parietal pleura is extensively involved, and in empyema necessitatis there is visible protrusion of a localized region of the chest wall with discoloration of the integument when rupture is imminent. The cardiac impulse is displaced toward the opposite side of the thorax, and the weight of the purulent effusion produces downward displacement of the diaphragm, with consequent downward displacement of the liver, which may produce bulging below the right costal margin. The subcutaneous veins of the thorax are apt to be tortuous over the lower thorax; and the diaphragmatic shadow is abolished upon the side of the effusion. In pulsating empyema there is systolic pulsation which is synchronous with the cardiac systole.

Palpation.—Vocal fremitus is abolished over the distribution of the purulent effusion, while above the level of the fluid, over the compressed lung, the fremitus is occasionally exaggerated. Pulsating empyema yields a palpable systolic pulsation, while in empyema necessitatis the edematous chest wall may pit upon pressure with the finger-tips.

Percussion.—There is dullness amounting to flatness over the purulent effusion, with Skodaic resonance above the level of the fluid, as in serofibrinous pleurisy. Percussion of the opposite lung yields a slightly hyperresonant note as the result of compensatory emphysema. Grocco's triangular area of paravertebral dullness is usually well-marked in empyema.

Auscultation.—The respiratory murmur is abolished over the purulent effusion, while it is puerile over the compressed lung above the level of the effusion. In children there is frequently blowing bronchial breathing above the level of the fluid.

Diagnosis.—Purulent pleurisy closely resembles serofibrinous pleurisy in its physical manifestations; but in empyema the disproportion between the two sides of the thorax is more marked; there is more apt to be intercostal bulging in empyema; and the visceral displacement in purulent pleurisy is more extreme. Edema of the thoracic wall points to purulent rather than to serofibrinous pleurisy. Moreover, empyema is attended by a greater degree of dyspnea than is serofibrinous effusion, the labored breathing frequently amounting to orthopnea. Purulent pleurisy is attended by less local pain than is serofibrinous pleurisy; but there are in empyema greater emaciation and signs of a generally septic state. Aspiration reveals the presence of purulent effusion in the pleural cavity. Baccelli's sign is occasionally of aid in the differential diagnosis.

CHRONIC ADHESIVE PLEURISY

Clinical Pathology.—Chronic adhesive pleurisy, or chronic plastic pleurisy is most frequently a sequence of serofibrinous pleurisy, more rarely of empyema, and occasionally it develops as a primary affection of the pleura.

Following the aspiration or absorption of a pleural effusion of the serofibrinous or purulent type, the surface of the pleura is covered with an exudate which is rich in fibrinous elements and moreover there are usually scattered areas in which the surface endothelium has desquamated, exposing the subjacent connective tissue basis of the pleural membrane. The surfaces of the visceral and the parietal pleuræ, coated as they are with fibrinous exudate, have a tendency to adhere to one another; and, the fibrinous exudate having undergone organization, the two surfaces become bound together by fibrous adhesions of variable extent. In extreme cases the surfaces may adhere throughout their entire extent, obliterating the potential pleural cavity, and restricting the respiratory excursion of the lung.

Physical Signs.—The physical signs of chronic adhesive pleurisy vary with the duration of the disease and with the extent of the pleural adhesions, varying from moderate dyspnea to extreme embarrassment of the respiratory function and striking thoracic deformity.

Inspection.—In cases of chronic adhesive pleurisy with moderate adhesions slight dyspnea may be the only sign of the disease, and even this sign may not be in evidence. But in the more extreme grade of the disease, in which numerous adhesions between the visceral and the parietal pleura bind the lung to the thoracic wall, local retraction or absolute immobilization of the corresponding side of the thorax is in evidence.

Palpation.—Upon palpation of the thorax pleural friction fremitus is demonstrable over the sites of pleural roughening and thickening. Palpation is apt to detect minor degrees of expansion and thoracic retraction in cases of moderate adhesions. In cases with considerable thickening of the pleura the intensity of vocal fremitus is diminished.

Percussion.—The percussion note is but little altered in cases of moderate pleural involvement; but the note is strikingly dull and the resistance to the pleximeter is marked in cases with obliteration of extensive portions of the potential pleural cavity. The respiratory excursion of the lung is greatly diminished, occasionally to complete immobilization, upon the side of the disease. The note upon percussion of the opposite lung in the extreme case is hyperresonant as a consequence of compensatory emphysema.

Auscultation.—In chronic adhesive pleurisy the respiratory murmur is very apt to be enfeebled over various areas or indeed in the extreme case over the entire side of the thorax. Over areas of pleural roughening the pleural friction sound is audible; and very frequently the râles of an associated chronic bronchitis are audible upon the side of the disease.

Diagnosis.—The diagnosis is made upon the presence of the pleural friction sound, the thoracic deformity and respiratory embarrassment, with a history of previous acute pleurisy.

HEMOTHORAX

Clinical Pathology.—The collection of blood in the pleural cavity may result from rupture of an aneurysm of one of the large intrathoracic arteries or from erosion of an intercostal vessel i the presence of pleural disease. Hemothorax also arises as result of trauma to the chest wall, notably after perforatin wounds from missiles or from a fractured rib. Gangrene of th lung may be responsible for the hemorrhage, or it may be a portion of a hemorrhagic diathesis. Rupture of aneurysm of th aorta usually produces hemothorax of the left pleural cavity.

The onset of hemothorax is usually very abrupt, and if one o the larger intrathoracic vessels is the source, it is usually rapidl fatal. In bleeding from an intercostal vessel the bleeding is ap to be slowly continuous for a variable time, after which it spontaneously ceases.

The amount of blood which is extravasated into the pleura cavity is variable. If infection does not occur, following arrest o the hemorrhage, complete absorption may occur; not, howeve without leaving pleural adhesions.

Physical Signs.—The initial signs of hemothorax are those o internal hemorrhage; namely, pallor, with dyspnea, rapid puls and collapse. Superimposed upon these signs are those of effusio in the pleural cavity; and exploratory puncture reveals the presence of sanguineous fluid.

CHYLOTHORAX

Clinical Pathology.—Effusion of chyle or chyliform fluid int the pleural cavity is only occasionally encountered. The fluid ma be derived from rupture of the thoracic duct or may be discharge by transudation from the lacteals. The thoracic duct may be ruptured by trauma to the thorax; or the duct may be obstructed b the pressure of an intrathoracic tumor. Again, a chylous ascite may discharge into the pleural cavity by way of the lymphatic Or, again, occlusion of the left subclavian vein, into which the thoracic duct empties its contents, may result in chylothorax.

Physical Signs.—The physical signs of chylothorax are essentially those of fluid in the pleural cavity. The nature of the effusion is determined by exploratory puncture of the pleura.

HYDROTHORAX

Clinical Pathology.—The transudation of serous, noninflammatory fluid into the pleural cavity occurs most frequently in connection with cardiac or renal disease, conditions which are not infrequently attended by general anasarca. Hydrothorax of cardiac origin is more pronounced upon the right side, and, indeed, the fluid transudation is occasionally confined entirely to the right pleural cavity. The predilection of this type of the disease for the right pleura is commonly ascribed to pressure which is presumably exerted upon the azygos veins by an enlarged heart.

The hydrothorax of renal origin is bilateral and the transudation does not attain to the degree which is commonly observed in connection with regurgitant cardiac lesions. Unilateral hydrothorax is occasionally caused by compression of the large veins by mediastinal tumor, aortic aneurysm, or pleural neoplasm.

In hydrothorax the fluid is clear and of slightly yellowish hue, with a specific gravity of 1.010 to 1.013, and is devoid of fibrin. Upon standing a slight sediment of flat, desquamated endothelial cells is demonstrable microscopically.

The pleura shows little alteration in uncomplicated cases of hydrothorax. In the presence of coincident pleurisy the membrane presents the characteristic changes of this disease, and the fluid contains a variable number of polynuclear leucocytes. Old pleural adhesions occasionally produce a condition of loculated or encysted hydrothorax.

Physical Signs.—The physical signs of hydrothorax are essentially those of effusion into the pleural cavity, without, however, the initial pleural friction sound of pleurisy. Occasionally limited entirely to the right pleural sac, the effusion is invariably greater upon the right side of the thorax. In cases of cardiac origin the murmurs of cardiac insufficiency are apt to be in evidence as well as definite changes in the cardiac outline; while in cases which are dependent upon renal disease signs referable to the kidneys are usually present. In the absence of general anasarca, moderate edema of the feet and ankles is frequently demonstrable in hydrothorax.

Exploratory puncture reveals the presence and the character of the fluid.

PNEUMOTHORAX (HYDRO-, HEMO-, OR PYO-PNEUMOTHORAX)

Clinical Pathology.—The accumulation of air or gas in the pleural cavity may result from trauma or may arise as a complicatio of pulmonary disease. Pneumothorax is not infrequently produced by perforating wounds of the chest wall by missiles or th sharp extremity of a fractured rib; and it may be similarly established by the rupture of empyema necessitatis.

Diseases of the lung by creating a communication between th bronchial system and the pleural cavity occasionally result i pneumothorax, as, for example, following the rupture of a tuberculous cavity which is situated immediately subjacent to th pleura. The development of the Bacillus aerogenes capsulatu in the pleural cavity causes primary pneumothorax.

In pneumothorax the pleural cavity contains air or gas, whic compresses the lung, which becomes shrunken and carnified. Th pleural cavity may be closed, with no communication with th exterior, constituting a closed pneumothorax; or it may possess communication with a bronchus or externally through the ches wall, constituting an open pneumothorax. Associated with th pneumothorax there is commonly a variable quantity of serou fluid, pus, or blood, constituting, respectively, hydro-, pyo-, o hemo-pneumothorax.

The liver or spleen is apt to be displaced downward, and th heart is displaced toward the opposite side of the thorax.

Physical Signs.—*Inspection.*—In pneumothorax the patient i usually dyspneic and the facial expression is anxious. The affected side of the thorax presents a variable degree of unilatera bulging, and the intercostal spaces are frequently obliterated Expansion upon the side of the disease is nil, and is in strikin contrast with the vicarious expansion of the opposite side of th thorax. The cardiac impulse is displaced laterally, toward th side of the sound lung. The patient as a rule prefers to lie upo the side of the pneumothorax in order to give the sound lun free play, though occasionally the dyspnea is so great that the upright posture is assumed. The diaphragmatic shadow is abolishe upon the side of the disease.

Palpation.—Vocal fremitus is abolished over the pneumothorax unless it is conducted to the surface of the thorax by pleural adhesions. The intercostal spaces offer increased resistance upo palpation with the finger-tips.

Percussion.—The percussion findings are variable in pneumothorax, depending upon the degree of tension of the air in the pleural cavity and the amount of fluid which is present therewith. When the pleural cavity contains a considerable quantity of fluid, percussion of the dependent portion of the cavity yields flatness, changing abruptly to a tympanitic note when the upper border of the fluid is passed. The limit of pulmonary resonance is extended upward above the clavicle, and in the absence of fluid downward over the areas of hepatic and splenic dullness as well. The coin test of Gairdner is readily elicited in pneumothorax, and when fluid is present, movable dullness is demonstrable.

Pneumothorax with patent bronchial communication frequently yields a cracked-pot sound upon forcible percussion, and occasionally Wintrich's change of note may be elicited. Also in these cases Biermer's phenomenon, an alteration in the pitch of the percussion sound with change in the patient's posture, may be elicited. The area of dullness of the heart is frequently diminished, percussion over the precordia yielding hyperresonance or tympany as a result of cardiac displacement. The dullness of the liver or the spleen is apt to extend to an abnormally low level.

Auscultation.—As a rule, the respiratory murmur is absent over a pneumothorax, though in some cases distant amphoric breathing is audible. The voice sounds are ringing and amphoric. Over the opposite side the breathing is puerile from compensatory emphysema. The metallic tinkle is audible in many cases, as well as the succussion sound upon suddenly jarring the patient. In cases with patent bronchial fistula the lung-fistula sound is apt to be elicited. The heart tones frequently possess a hollow, echoing sound, due to the proximity of the air in the pleural cavity.

Diagnosis. The unilateral bulging, with suppressed or absent breath sounds, tympanitic percussion note, the falling-drop sound and the succussion sound, with cardiac and visceral displacement, constitute a characteristic picture.

The differential points between pneumothorax, pleural effusion, and hydrothorax, have been discussed in a previous section (see pp. [illegible])

SECTION IV

PHYSICAL EXAMINATION OF THE CIRCULATOR ORGANS

CHAPTER XII

CLINICAL ANATOMY

THE HEART

The heart, the great muscular pump by which the column o **blood is propelled through** the vessels, is a roughly conical orga **situated obliquely in the** middle mediastinum between the lung **whose anterior borders** partially overlap its ventral surfac **The heart presents for** examination a base, which is directed upward **and toward the right**, an apex, which is directed downwar **and toward the left**, and three borders; namely, right, left, an **inferior. The heart** does not occupy the midpoint of the thoraci **cavity; but projects** farther to the left of the median line than t **the right. Approximately** one-third of the organ lies in the righ **half of the** thoracic cavity, and two-thirds to the left of th **median line.**

The heart is a hollow muscular organ, comprising four chambers **or cavities**, an *auricle* and a *ventricle* upon either side, th **cavities of the** one side being separated from those of the opposit **side by a muscular** septum. Each auricle, on the contrary, communicates **with the** corresponding ventricle through the auriculoventricular **orifice.** The left auricle and ventricle contain arteri **blood**, while the right auricle and ventricle contain venous bloo

The external surface of the heart presents definite markings, i **the form of** transverse and longitudinal furrows, which accuratel **indicate** the internal subdivisions of the organ. The auriculoventricular **groove** encircles the heart transversely at a point somewhat **nearer** the base than the apex of the heart. This groov **corresponds** upon the surface of the heart to the site of the auriculoventricular **septa**, and lodges the coronary arteries. The interventricular **furrows** pass downward from the auriculoventricula **furrow** in the direction of the long axis of the heart, upon it anterior and posterior surfaces, respectively, terminating at the inferior sharp margin of the heart, which they reach a little distance to the right of the apex.

The cardiac wall is formed by bundles of specialized involuntary muscular fibers, the cardiac muscle, or *myocardium,* the bundles of which have a characteristic arrangement. The muscular bundles take origin from the fibrous rings which guard the auriculoventricular and aortic orifices. In the auricles the bundles are arranged in two layers, a superficial and a deep lamina, the latter composed of looped and annular fibers.

In the ventricles the bundles are likewise disposed in superficial and deep strata. The deep bundles encircle the ventricles, while the superficial fibers pursue a spiral course, coiling inward at the apex in the form of a whorl or vortex.

The myocardium is not of uniform thickness. The auricular walls are much thinner than are those of the ventricles; and the right ventricle does not attain to the same thickness as does the corresponding ventricle of the opposite side. The cavities of the heart are lined by a serous membrane, the *endocardium,* which is reflected over the cusps of the cardiac valves. The external surface of the myocardium is clothed by a similar serous membrane, the *epicardium,* which constitutes a portion of the visceral layer of the pericardium.

The *right auricle* consists of two portions: a principal cavity, the *sinus venosus,* or *atrium;* and an anterior recess, the *auricular appendix.* The walls of the right auricle represent the thinnest portion of the myocardium, and they readily dilate when an increased load is thrown upon the right side of the heart by an obstacle to the circulation of the blood through the pulmonary circuit. The interauricular septum presents a depression upon the inner wall of the auricle, the *fossa ovalis,* corresponding to the foramen ovale of the fetus. In certain cases of congenital heart disease, and indeed in 14 per cent of adults, this foramen does not close completely, its patency often producing obscure physical signs. The capacity of the right auricle averages sixty cubic centimeters, slightly exceeding the capacity of the left auricle. The right auricle communicates with the right ventricle by the auriculoventricular orifice, which is guarded by the tricuspid valve; while it receives venous blood from the large superior and inferior venæ cavæ, the former returning blood from the head, neck, and upper extremities; the latter returning blood to the heart from the trunk and lower extremities.

The *left auricle,* smaller than the right auricle, has thicker wal than the latter and hence its contractile power is greater. Lik the right auricle, it is composed of an atrium and an auricula appendix. The left auricle communicates with the left ventricl by the bicuspid or mitral valve and it receives blood from the lung through the large pulmonary veins. These large vessels usuall empty their contents by four orifices in the base of the auricle; bu not infrequently these orifices are reduced to three, the two lef pulmonary veins terminating in a common trunk.

The *right ventricle,* triangular in contour, extends from th right auricle to the lower sharp border of the heart, reaching almos to the apex of the organ. Its anterior surface is convex and constitutes the major portion of the ventral aspect of the heart, th portion which is in relation to the anterior chest wall in the interva between the incisura cardiaca of the left lung and the left sterna border. The posterior surface of the ventricle is flattened and rest upon the central tendon of the diaphragm. The right ventricl has a capacity of about ninety cubic centimeters, and its wall are not as thick as are those of the left ventricle. The ventricular wall is thinnest in the region of the superior and interna angle of the ventricle, the *conus arteriosus,* which overlies th opening of the pulmonary artery. In addition to the openin of the pulmonary artery, which is guarded by the semilunar valve the ventricle communicates with the right auricle by the righ auriculoventricular orifice, which is guarded by the tricuspid valv

The internal surface of the ventricle presents numerous muscula columns, *columnæ carneæ,* projecting from the myocardium an invested with endocardium. Three, or it may prove to be four, o these muscular columns are very well developed and constitute th *papillary muscles,* which are connected with the margins of th cusps of the tricuspid valve by delicate tendinous cords, *chord tendineæ.*

The *left ventricle,* longer, thicker, and more conical in shap than the right ventricle, forms only a limited portion of the anterior surface, but the major portion of the posterior surface of th heart. The inferior extremity of the left ventricle alone forms th apex of the heart. The capacity of the left ventricle varies fron eighty to one hundred cubic centimeters, and its walls are thicke than any other portion of the myocardium.

The interior of the left ventricle is similar anatomically to tha of the right ventricle. The ventricular cavity communicates witl the left auricle by the left auriculoventricular orifice, which i guarded by the bicuspid or mitral valve; and anteriorly and to the right of this valve it presents the aortic orifice, guarded by the semilunar valves. The right cusp of the mitral valve intervenes between the auriculoventricular and the aortic orifices; and, in the presence of aortic insufficiency, this cusp is apt to become the target of two streams of blood entering the left ventricle in opposite directions, the one from the left auricle and the other regurgitating from the aorta, and to be thrown into vibration with the production of a murmur, the murmur of Austin Flint, which will be described in a subsequent paragraph.

THE CARDIAC VALVES

The left auriculoventricular, *bicuspid*, or *mitral valve*, intervening between the left auricle and left ventricle, consists of a fibrous ring supporting two semilunar segments, or cusps. The concavity of each of these cusps is directed toward the ventricle; and when, during ventricular systole their free margins are approximated, the auriculoventricular orifice is securely closed, and regurgitation of blood from the ventricle into the auricle is effectually prevented. Inversion of the valve segments during ventricular systole is prevented by traction upon their free borders by the papillary muscles through the medium of the chordæ tendineæ.

The right auriculoventricular, or *tricuspid valve*, comprises three segments or cusps, supported at their bases by a fibrous ring. Their action is in all respects identical with that of the mitral valve, and their inversion during ventricular systole is similarly prevented by contractions of the papillary muscles with their chordæ tendineæ.

The *aortic valve* consists of a fibrous ring supporting three semilunar cusps, with their concave surfaces directed toward the lumen of the aorta. This valve is devoid of chordæ tendineæ and papillary muscles; but each segment is reinforced by a thin cartilaginous plate, the corpus arantii. The valve is closed by the force of the blood column which is ejected into the aorta during the systole of the left ventricle. Hence, the closure of this valve is diastolic, whereas that of the mitral and of the tricuspid valves is systolic in time. Opposite each segment of the aortic valve the wall of the aorta presents a small pouch or dilatation, the *sinus of Valsalva*, from two of which arise the coronary arteries, which nourish the myocardium.

The *pulmonary valve*, which intervenes between the right ventricle and the pulmonary artery, is similar anatomically to the aortic valve and its closure is effected in the same manner.

The function of the cardiac valves is to maintain a constant flow of blood in one direction. Owing to the delicacy of their structure, they become ready targets for infectious material which may gain access to the blood stream. As a result of such infection, structural changes, resulting in permanent deformity of the cusps, are apt to ensue, thus destroying the integrity of one or more of the valves. Such changes are inevitably followed by the sequence of events described under the effects of chronic valvular disease. Or, in the absence of infection and structural alteration in the valve segments, in the presence of malnutrition or excessive physical strain the fibrous rings at the orifices may stretch, with the result that the normal segments can no longer close the abnormally large orifice. In certain instances congenital deformities of one or more of the cardiac valves are the cause of imbalance of the circulation.

THE BUNDLE OF HIS

The only direct connection between the auricles and ventricles is the auriculoventricular bundle of His, which originates in the auriculoventricular node of Tawara beneath the epicardium in the base of the interauricular septum. Passing downward in the interauricular septum to the auriculoventricular junction, the bundle crosses the junctional tissues at this point; and, continuing its course in the interventricular septum, divides to form two principal branches, right and left, respectively. These two trunks pass downward upon either side of the interventricular septum, splitting into smaller and smaller divisions as the cardiac apex is approached. Eventually the finer terminals of the bundle reach the bases of the papillary muscles, and beneath the endocardium are distributed generally to the entire ventricular musculature.

It is generally conceded that the bundle of His transmits the impulse which results in systolic contraction from the auricles to the ventricles, and that disease affecting the integrity of these fibers plays an important part on the phenomena of heart-block and Stokes-Adams' disease.

THE PERICARDIUM

The pericardium is a conical seromembranous sac situated between the lungs in the middle mediastinum, and enveloping the heart and the great vessels arising from its base. With its apex directed upward and its base moored to the central tendon of the diaphragm by areolar tissue, the pericardium lies behind the sternum and the costal cartillages of the third, fourth, fifth, sixth, and seventh ribs of the left side. Anteriorly the pericardium is separated from the chest wall in the greater portion of its extent by the anterior borders of the lungs; but a small portion of the sac is in direct relation with the thoracic wall in the interval between the left sternal border and the anterior border of the left lung in the fourth and fifth intercostal spaces. The posterior surface of the sac is in contact with the bronchi, the esophagus, and the thoracic aorta.

In the presence of inflammatory affections of the structures with which the pericardium is in contact, adhesions are apt to form, causing displacement of the sac with its contents. Or, adhesions may form between the sac and the chest wall, resulting in retraction of the surface of the thorax.

The pericardium is composed of an outer fibrous coat, which is prolonged upward upon the great vessels which arise within the sac, finally to blend with the deep cervical fascia; and of an internal serous coat, which is reflected on to the heart as the epicardium. This layer secretes a small amount of serous fluid, enabling the parietal and visceral layers of the pericardium to glide noiselessly over each other during the cardiac contractions. In the presence of inflammation of the membrane, however, the membranes become dry and covered with exudate, with the production of a to-and-fro friction sound, corresponding fairly closely with the cardiac systole and diastole.

In pericarditis with effusion the fluid is prone to collect on either side of the heart and below it, causing a gradual elevation and a final obliteration of the visible cardiac impulse. The pericardium and heart are separated by the diaphragm from the left lobe of the liver, and upon the extreme left, corresponding to the position of the apex of the heart, from the stomach. Hence in pericardial effusions the fluid gravitates toward the left, as the diaphragm yields more readily to pressure on this side than it does upon the right, where it is in relation to the left lobe of the liver.

THE AORTA

The aorta, the principal arterial trunk of the greater or general circulation, arises from the aortic orifice at the superior and posterior portion of the left ventricle near the center of the heart at the level of the third left costal cartilage. The course of the thoracic aorta roughly resembles a shepherd's crook; and the vessel is divided into three portions, the ascending aorta, the aortic arch, and the descending aorta. The ascending aorta, which is enveloped by the apical portion of the pericardium, is approximately one and one-eighth inches in diameter, presenting near its origin the sinuses of Valsalva. The ascending aorta is two inches long, and at the upper border of the second right costal cartilage becomes continuous with the arch of the aorta. The ascending aorta occupies a position one-fourth inch behind the posterior surface of the sternum, separated from this bone by the enveloping pericardium and the right pleural sac. Near its termination the ascending aorta presents a dilatation, the *sinus maximus* which renders the lumen of the vessel at this point fusiform or oval. An aneurysm of the vessel at this point upon rupture discharges its blood directly into the pericardium with speedily fatal result. Prior to rupture an aneurysm of this portion of the aorta is prone to compress the right auricle, superior vena cava, or pulmonary artery, leading to embarrassment of the circulation and engorgement of the superficial veins of the neck.

The *aortic arch* commences at the level of the upper border of the second right costal cartilage and arches upward, backward, and to the left behind the manubrium sterni and in front of the trachea just above its bifurcation. Thence the vessel crosses over the left bronchus and passes downward to the left side of the body of the fourth thoracic vertebra, where it becomes continuous with the descending thoracic aorta. In the superior mediastinum three large arterial trunks, the innominate, the left common carotid, and the left subclavian, spring from the convexity of the arch; while by its lower concave aspect it is connected with the pulmonary artery by a fibrous cord, the ligamentum arteriosum, the remnant of the obliterated ductus arteriosus of the fetus.

Aneurysmal dilatation of the posterior wall of the aortic arch is apt to compress the trachea, producing cough, stridor, hemoptysis or fatal hemorrhage in the event of rupture. Pressure may also be exerted upon the esophagus with the production of dysphagia or upon the thoracic duct, resulting in lymph stasis or chylothorax. Similarly aneurysm of the anterior aspect of the arch may cause sternal protrusion with boring pain beneath the sternum. Through irritation of the left recurrent laryngeal nerve, which winds around this portion of the vessel, laryngeal symptoms may be induced, and through disturbance of the sympathetic nervous system pupillary changes may occur.

The *descending aorta*, commencing at the left side of th border of the fourth thoracic vertebra, descends in the deep portion of the posterior mediastinum to the aortic orifice of the diaphragm, through which it passes in front of the body of the twelfth thoracic vertebra. Aneurysm of this portion of the aorta, which is not an uncommon disease, usually extends backward along the left side of the vertebral column, where it causes boring pain and absorption of the bodies of the vertebræ, resulting in scoliosis or lordosis. When developing upon the anterior wall of the vessel, an aneurysm exerts pressure upon the pericardium and heart, pushing these structures before it, and giving rise to physical signs which suggest cardiac hypertrophy or pericarditis with effusion. In either situation rupture may occur into the mediastinum or pleural cavity with fatal termination.

Congenital abnormalities of the aorta are not common, though occasionally the aortic valve may present an abnormality. This may consist of adhesions between the borders of the valve cusps, or fenestration of the segments; or in rarer instances the orifice may be represented by a narrow slit, the button-hole orifice of Corrigan.

THE PULMONARY ARTERY

The pulmonary artery, springing from the base of the right ventricle at the conus arteriosus, lies anterior to all of the great vessels which are connected with the base of the heart. A short wide trunk, two inches in length and embraced by the right and left auricular appendages, the pulmonary artery passes upward and backward to reach the concavity of the aortic arch, where, in front of the bifurcation of the trachea, at the level of the fourth dorsal vertebra, it divides to form the right and left pulmonary arteries, which enter the roots of their respective lungs.

Opening into the left auricle are the four *pulmonary veins*, which return arterial blood from the lungs to the left heart, while emptying into the right auricle are the *superior vena cava*, returning venous blood from the upper portion of the general circulation, and the *inferior vena cava*, returning venous blood from the abdominal cavity and lower extremities.

TOPOGRAPHICAL ANATOMY

The Heart.—The *base* of the heart, formed by the right and left auricles, corresponds to a line crossing the sternum obliquely from the lower border of the second left costal cartilage at a point one-half inch to the left of its junction with the sternum to the upper border of the third right costal cartilage at a point one inch beyc its sternal junction.

The ***right border*** of the heart, formed by the right auricle, cor sponds to a curved line, with its convexity directed toward right, extending from the upper border of the third right cos cartilage one inch from its junction with the sternum, to the si right chondrosternal articulation.

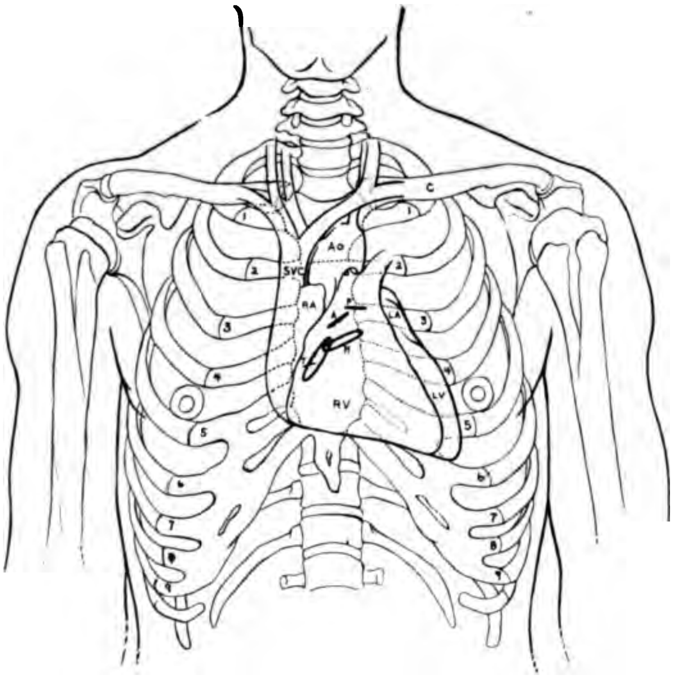


Fig. 117.—Relation of chambers of unopened heart to anterior thoracic wall. left auricle; *LV*, left ventricle; *RA*, right auricle; *RV*, right ventricle; *P*, pulm valve; *A*, aortic valve; *M*, mitral valve; *T*, tricuspid valve; *AO*, aorta; *SVC*, supe vena cava.

The *inferior border* of the heart, formed almost entirely by right ventricle, and to a minor extent by the left ventricle, cor sponds to a line drawn from the sixth right chondrosternal arti lation to the site of the normal cardiac impulse in the fifth l intercostal space one-half inch internal to the midclavicular li

The *left border* of the heart, formed by the left ventricle, represented by a curved line drawn with its convexity direc upward and toward the left, from the fifth left intercostal sp one-half inch internal to the midclavicular line to the lower bor of the second left costal cartilage, one-half inch to the left of articulation with the sternum.

The *cardiac valves* all lie within a small ellipse extending from the third left chondrosternal articulation to the junction of the sixth right costal cartilage with the sternum. Within this elliptical area the *pulmonary valve* lies behind the third left chondrosternal articulation at the level of the upper border of the third costal cartilage. The *aortic valve* occupies a position behind the sternum a little distance to the right of the pulmonary valve at the level of the third intercostal space. The *mitral valve* lies behind the left sternal border at the level of the fourth costal cartilage. The *tricuspid valve* occupies a position behind the body of the sternum corresponding to the central point of a line drawn obliquely across the midsternal line from the third left to the sixth right chondrosternal articulation. In relation to the anterior wall of the thorax, the pulmonary valve is most superficial; the tricuspid is next in depth; the aortic more deeply placed than the tricuspid; while the mitral valve occupies the position most remote from the chest wall. This area overlies the anatomic site of the valves, and is not the area in which the sounds produced at the several valves are best appreciated acoustically.

The Aorta.—The ascending portion of the aorta is represented on the surface of the thorax by a broad line drawn from the third left chondrosternal junction to the upper border of the second right costal cartilage at its junction with the sternum. From this point the arch of the aorta takes a course backward and toward the left behind the manubrium sterni, its convexity ascending to a point one inch below the episternal notch, its concavity corresponding to the level of the angle of Louis.

The *pulmonary artery* corresponds to a broad line drawn from the second left intercostal space to the upper border or the second left costal cartilage, the point of bifurcation of the artery to form its two primary branches.

The Precordia.—The term precordia is applied to the area of the surface of the thorax which overlies the heart and pericardium. The name does not refer merely to the limited region in which the heart is directly in apposition with the thoracic wall, but also to the region in which the anterior borders of the lungs overlap the pericardium and heart. The region embraces the areas of cardiac dullness and cardiac flatness, to be described in a subsequent section. The precordia presents sharp lines of demarkation superiorly and upon the left; but upon the right side and inferiorly it is continuous with the areas of hepatic dullness and flatness.

CHAPTER XIII

INSPECTION AND PALPATION

Object and Technic.—In the examination of the circulat organs inspection and palpation may be combined with adv tage, local findings upon inspection being corroborated and c firmed by palpation of the area in question.

Before proceeding to the inspection of the precordia and perior thoracic regions, the examiner should make a general spection of the body as a whole during which he notes the prese of dyspnea, cyanosis, clubbing of the fingers, edema or anasar and during this examination the color of the integument sho receive due attention. The pallor of the skin attending the l stages of chronic valvular lesions associated with arterial hy tension and deficient peripheral circulation presents a striki contrast with the cutaneous flushing which attends arterial pertension, aortic regurgitation, and vasodilatation due to ac febrile affections.

General inspection should be followed by local inspection the thorax and upper abdomen for the purpose of detecting site and character of the cardiac impulse, undue prominence recession of the precordia, and extra-apical pulsations of cardi arterial, or venous origin, as well as the presence of capill pulsation in Corrigan's disease.

During the examination the subject should occupy the se prone posture if this position is not contraindicated by distre ing subjective symptoms. On account of the urgent dyspr which attends many cases of cardiovascular disease, the exami tion must frequently be conducted with the patient in the sitti posture. Again, in the study of certain variations in the char ter of the cardiac impulse it is desirable to have the patient be the trunk slightly forward. Moreover, in testing the lateral n bility of this impulse the subject is placed alternately in the ri and left lateral decubitus.

Pulsations which are observed during inspection should further studied and defined by careful palpation of the thor or abdomen. Likewise, palpation may reveal the presence tactile vibration, fremitus or thrills, over the precordia or at several valve areas.

PRECORDIAL BULGING

Bulging of the precordia arises from endocardiac and extracardiac causes. In general, undue prominence of the precordia which is referable to endocardiac lesions is restricted to the area between the third and seventh ribs and between the left sternal border and the left midclavicular line; but this is not an invariable rule, as pericardial effusion developing in the young subject may produce bulging extending from nipple to nipple.

The degree of precordial bulging is influenced largely by the age and sex of the subject. Congenital heart disease and pericarditis with effusion developing early in life produce extensive bulging of the precordia. Excessive hypertrophy of the heart in the child and in the female subject is attended by precordial bulging, but in the case of the more rigid thorax of the adult male subject bulging under these circumstances is scarcely ever to be noted. Aneurysm of the aortic arch produces bulging of the superior portion of the precordial space, the prominence in this instance extending well beyond the limits of the precordia.

Precordial bulging of extracardiac origin arises as a result of the progressive growth of a neoplasm of the mediastinal structures, lung, pleura or chest wall, as a local manifestation of a localized pleural effusion or empyema necessitatis, or as a result of caries of the sternum.

PRECORDIAL RETRACTION

Undue recession of the precordia is frequently the result of the traction of pericardial or pleural adhesions. A not infrequent cause of retraction in this region is fibrosis of the anterior border of the left lung, arising during the course of fibroid phthisis or chronic interstitial pneumonia. Precordial retraction is likewise occasionally noted in which the underlying cause is extensive cavitation of the left lung during the course of chronic ulcerative phthisis. Similarly, recession of the lower portion of the precordia constitutes the funnel-chest, whether the result of occupation or occurring as a congenital defect.

THE CARDIAC IMPULSE (APEX BEAT).

With each systole of the ventricles there is to be noted a circumscribed elevation of the thoracic wall which is superjacent to the inferior portion of the right ventricle, constituting the cardiac impulse, or apex beat of the heart. The cardiac impulse usually visible, and in the cases in which it is not to be detec upon inspection it is usually readily localized by palpation. is to be noted, however, that in the case of the deep-chested s ject with voluminous anterior pulmonary borders, as well as the obese subject and in those cases in which the right ventricu apex occupies a position directly behind a rib, the impulse sometimes neither visible nor palpable.

In the normal adult male subject the cardiac impulse is lo

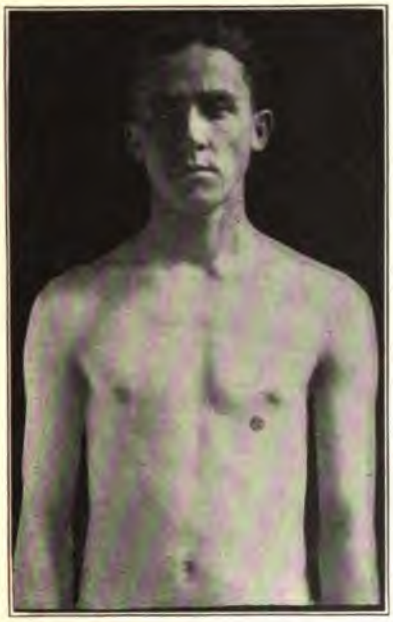


Fig. 118-*A*.—Site of normal cardiac impulse.

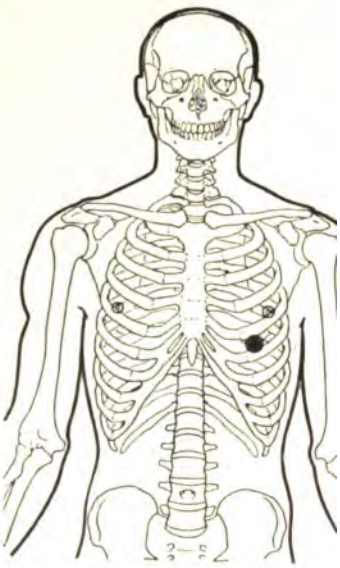


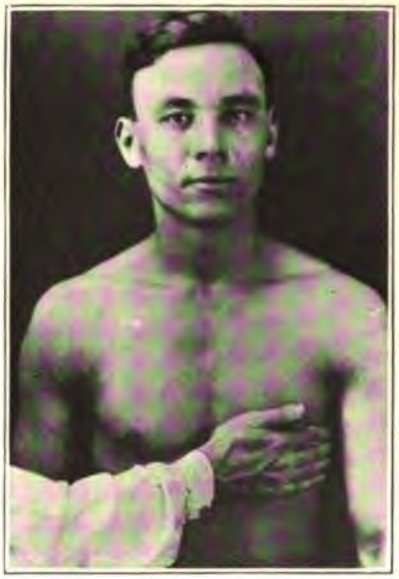
Fig. 118-*B*.—Site of normal cardiac i

ized in the fifth left intercostal space one-half inch internal to midclavicular line. Measured from the median line of the tho the impulse is situated 3.5 inches or 8.75 centimeters to the of the midsternal line.

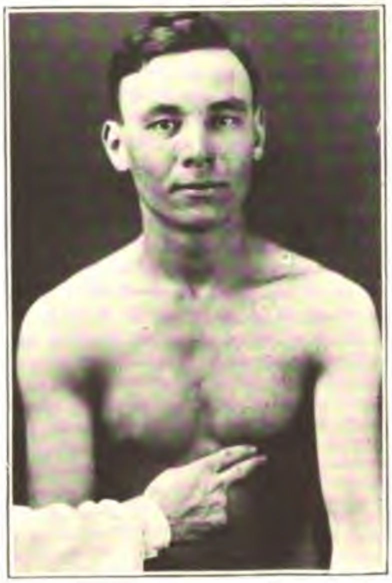
The site of the cardiac impulse is influenced by the age of subject, the structure of the thorax, the movements of resp tion, the attitude of the body, and occasionally by violent e tional excitement or physical effort.

During infancy and early childhood, as a consequence of relative shortness of the thoracic cavity and the relative incre in the vertical diameter of the abdominal cavity obtaining at

he cardiac impulse is not infrequentl
intercostal space. Also, in this class
ie relatively large dimensions of the



119.—Palpation of cardiac impulse (first maneuv



120.—Palpation of cardiac impulse (second mane time of life, the impulse often occupies a position external to th midclavicular line. In the aged subject, on the contrary, partiall as a result of hardening and straightening of the thoracic aort and partially as a result of ptosis of the abdominal viscera an diaphragm, the heart commonly occupies an abnormally low p sition in the thoracic cavity, and the cardiac impulse is encou tered in the sixth or even in the seventh intercostal space.

In many instances the structure of the thorax influences th site of the cardiac impulse. In the subject with an unduly sho thorax the impulse is not infrequently visible in the fourth inte space; whereas, in the subject of the long expiratory type thorax it is not uncommon to find the impulse situated in the sixt intercostal space. Moreover, thoracic deformities due to osseo disease or to rickets are commonly attended by displacement the cardiac impulse in the absence of disease of the heart.

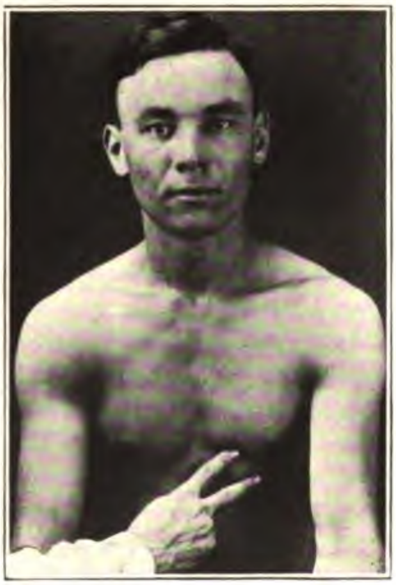


Fig. 121.—Palpation of cardiac impulse (third maneuver).

During forced inspiration the cardiac impulse is occasionall displaced downward to the extent of one interspace, and it a cends to the fifth interspace during expiration. The excursio of the lungs during tranquil respiration, however, produces n appreciable displacement of the cardiac impulse. In the pre ence of marked stenosis of the larger bronchi it occasionally ha pens that during inspiration the diaphragm not only does not descend, but it ascends into the thoracic cavity more forcibly than is the case during normal expiration. In this event there is observed a reversal of the ordinary sequence of events, with upward displacement of the apex beat during forced inspiration.

As a result of active physical effort, and occasionally in the train of violent emotional excitement, the cardiac impulse is increased in extent and assumes a position nearer the midclavicular line than is the case in the normal subject in repose.

The attitude of the body exercises a striking influence upon the site of the cardiac impulse in the normal subject. When the subject assumes the left lateral decubitus, as a consequence of the normal lateral mobility of the heart, the impulse is displaced toward the left axillary line to the extent of one to two inches. Upon assuming the right lateral posture the apex beat is displaced toward the sternal border, but the mobility of the organ in this direction never equals its movement toward the left side of the thorax. Increase in the lateral mobility of the heart is noted in connection with ptosis of the organ, whereas limitation of the normal cardiac mobility is usually indicative of the presence of pericardial adhesions. In whatever site the cardiac impulse is detected, a correct localization of the area of impulse affords very accurate information as to the position of the heart within the thoracic cavity.

The normal cardiac impulse covers an area corresponding to the width of the intercostal space in which it is localized, with a lateral dimension of approximately one inch. The impulse is more extensive when the subject assumes the sitting posture or when the trunk is inclined forward than when the semiprone or recumbent posture is assumed. In the normal subject in the area of the cardiac impulse with each ventricular systole the integument is elevated to a plane corresponding with the adjacent ribs. Elevation of the impulse above the plane of the adjacent ribs is indicative of accentuation of the cardiac impulse.

In the localization of the cardiac impulse by palpation, the palm of the hand should first be applied firmly and evenly to the area in which the impulse should be encountered. When the site of the impulse is thus noted, the more sensitive finger-tips should be applied to the area; and, by separating the fingers, the extent of the apex beat is accurately estimated.

DISPLACEMENT OF THE CARDIAC IMPULSE

Aside from the physiological displacement to which the cardia impulse is subject, various abnormal displacements of the impuls may be encountered, arising from endocardial and exocardia causes. Under these circumstances the direction and degree o apical displacement not infrequently furnish a clue to the caus of the displacement.

Upward Displacement of the cardiac impulse is noted in th presence of cardiac atrophy, in the presence of diaphragmati hernia, and when the diaphragm is elevated as a result of increased intraabdominal tension. In the presence of fibroid retraction of the anterior border of the left lung the impulse is elevated and it is commonly visible over an extensive area along the lef sternal border. The pressure of an extensive effusion into th right pleural sac occasionally causes the liver to rotate about it axis in such a manner as to tilt the narrow left lobe of the live upward, resulting in elevation of the superjacent heart and displacement of the cardiac impulse upward and toward the left.

Downward Displacement of the cardiac impulse occurs as consequence of the progressive development of an aneurysm o the aortic arch or of a mediastinal tumor which presses upon th base of the heart. A similar displacement frequently attend hypertrophic emphysema, partially as a result of pressure exerte upon the heart by the voluminous anterior borders of the lungs i this disease, and partially as a result of the inspiratory positio of the thorax peculiar to this disease, in which the ribs tend t move upward over the heart, simulating a downward displacement of the heart.

Extensive pericardial effusion, prior to the obliteration of th cardiac impulse, is apt to be attended by depression of the diaphragm, in which event the cardiac impulse is displaced downward.

In addition to these exocardial pulsion and traction displacements, the cardiac impulse is displaced downward and toward th left in left ventricular hypertrophy and dilatation, the impuls under these circumstances not infrequently occupying the sevent or eighth interspace external to the left midclavicular line.

Displacement to the Left.—In addition to the left lateral displacement of the cardiac impulse which attends left ventricula hypertrophy and dilatation, a further important cause of suc displacement is right ventricular hypertrophy and dilatation, i which the apex beat is frequently localized external to the left midclavicular line. Horizontal displacement of the impulse toward the left likewise occurs as a consequence of the pressure exerted by the progressive accumulation of fluid or gas in the right pleural cavity, and as a result of the traction of adhesions between the pericardium and left pleura.

Fibroid retraction of the anterior border of the left lung results in left lateral displacement and elevation of the cardiac impulse; and a similar displacement of the impulse attends hepatic enlargement and gastric distention. In the presence of pericardial effusion of moderate degree the apex beat is frequently elevated and displaced toward the left.

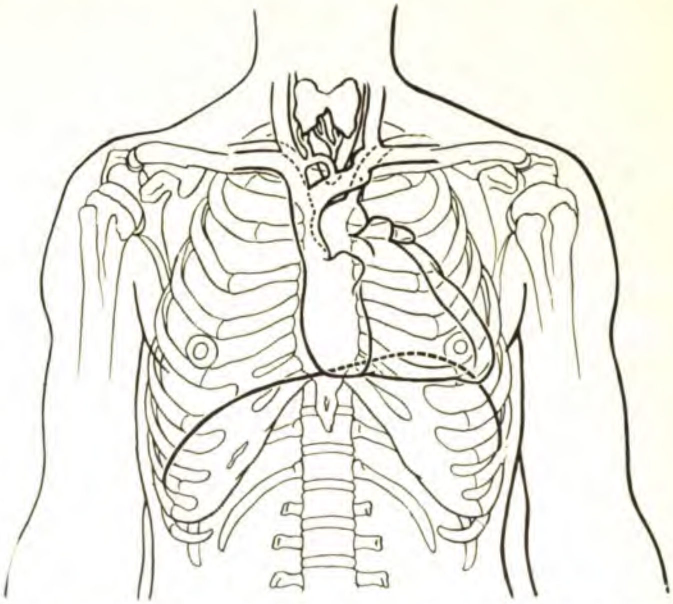


Fig. 122.—Illustrating moderate displacement of the heart towards the left in compensatory emphysema of the right lung, with elevation of the left vault of the diaphragm and displacement toward the left of the structures in the median line of the neck.

Displacement to the Right.—The cardiac impulse is displaced toward the right by the pressure of fluid or gas in the left pleural cavity, and it is drawn to that side by the traction of right pleuropericardial adhesions. Compensatory emphysema of the left lung, when sufficient in degree, may displace the impulse toward the right. Similarly, the progressive development of a neoplasm of the left lung is capable of causing apical displacement toward the right side of the thorax. In the presence of congenital transposition of the thoracic viscera the cardiac impulse is encou tered in the fifth right intercostal space.

EXTENT OF THE CARDIAC IMPULSE

Normally occupying an area approximately one inch in widt the cardiac impulse in the presence of disease presents marke variations in the extent of the visible or palpable pulsation. U der these circumstances the impulse may occupy an abnormall extensive area; it may be diminished in extent; or it may totally absent upon physical examination of the subject.

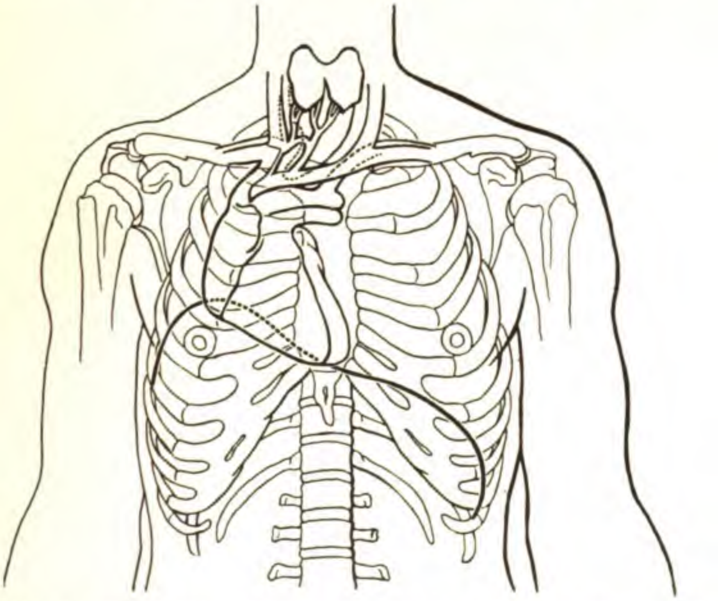


Fig. 123.—Illustrating cardiac displacement toward the right in compensatory emph sema of the left lung, which is attended by depression of the diaphragm upon the l side and by displacement of the structures of the median line of the neck toward right side.

Increased Extent.—The area occupied by the cardiac impul is increased in extent during emotional excitement and followin violent physical effort without possessing untoward significan Similarly, in the case of the obese patient and in the female su ject the impulse is imparted to the soft structures in such a diffu manner that an extensive impulse is generated, which is acc rately defined with considerable difficulty. In the case of t resilient chest wall of the child, again, the impulse is visible palpable over a more extensive area than in the adult subje

Extension of the area of impulse during childhood should not be attributed to hypertrophy of the ventricle, therefore, in the absence of coincident displacement of the impulse from its normal site. A further exocardial cause of increase in the area of the cardiac impulse is to be found in retraction of the anterior border of the left lung, thus exposing a broad area of the right ventricle to the anterior thoracic wall.

Increase in the area of the cardiac impulse of endocardial origin occurs in cardiac overaction due to disturbances of innervation or acute fevers, and in the presence of ventricular hypertrophy and dilatation. In the latter event the impulse is, moreover, displaced from its normal site.

Diminished Extent.—With the increasing rigidity of the bony thorax incident to advancing age, the cardiac impulse becomes visible over a more restricted area of the thorax, and it may become invisible and impalpable in the aged subject. Similarly, in deep-chested subjects there is apt to be a very slight impulse, while in the subject of hypertrophic emphysema the impulse is restricted in its area or is absent. In the more extreme grades of ventricular dilatation, moreover, instead of occupying an abnormally wide area upon the surface of the thorax, the cardiac impulse may be restricted in extent or abolished.

FORCE OF THE CARDIAC IMPULSE

As the cardiac impulse in the presence of disease of the heart and adjacent thoracic viscera presents variations in its area of impulse, so also under somewhat similar circumstances the force of the impulse is altered, with or without possessing untoward significance.

Increased Force.—During periods of emotional excitement and after excessive physical effort there is a transient increase in the force of the cardiac impulse. A similar temporary accentuation of the impulse follows the ingestion of stimulants and occurs during the course of acute febrile diseases.

In the presence of hypertrophy of the ventricles, in addition to its increased extent, the cardiac impulse possesses undue force or lifting power upon palpation. In this connection it is to be borne in mind that ventricular hypertrophy is constantly attended by displacement of the cardiac impulse. During the evolution and course of acute myocarditis the force of the cardiac impulse is appreciably increased.

Diminished Force.—In the presence of edema of the thorac wall the force of the cardiac impulse, as appreciated by palp tion, is distinctly diminished without reference to the state of tl myocardium. Hypertrophic emphysema is likewise producti of diminution of the palpable intensity of the cardiac impuls even though this disease is commonly attended by modera hypertrophy of the right ventricle.

Diminution in the force of the impulse is noted when cardi dilatation supervenes upon hypertrophy, in the presence of my cardial degeneration, and in pericarditis with effusion of mode ate degree. In pericarditis, however, the impulse is apt to b come visible or palpable when the trunk is inclined forward, onl to disappear when the upright posture is resumed.

DOUBLE IMPULSE

Occasionally the area of the cardiac impulse presents two di tinct pulsations to a single arterial pulse as recorded in the radi artery. This discrepancy between the cardiac impulse and tl arterial pulse may be due to the fact that the ventricles contra asynchronously, or to the fact that ventricular systole occurs wit a varying intensity, not every systole of the left ventricle po sessing sufficient force to produce an arterial pulse.

SYSTOLIC RECESSION

During the forcible contraction of the heart in the presence left ventricular hypertrophy, and with less constancy in the pre ence of cardiac dilatation, there is occasionally to be noted systolic recession of the area immediately adjacent to the site the cardiac impulse. The recession in this case is to be attribute to the action of atmospheric pressure, and should not be co fused with the more extensive traction recession of the thoraci wall, which occurs with chronic adhesive pericarditis.

EXTRA-APICAL PULSATION

Abnormal areas of pulsation along the borders of the preco dia or in more remote regions are noted during the course o cardiovascular disease; and they possess a variable significanc depending upon their sites and their relations to the events o the cardiac cycle.

Pulsation at the Base.—Visible pulsation at the base of th heart, which is synchronous with ventricular systole, is indicative in the vast majority of cases of aneurysm of the transverse portion of the aortic arch. As a rule, when visible pulsation is present in this region the aneurysm has eroded the sternum, and is attended by pain of a dull, boring character. With less constancy pulsation at the base is referable to extreme cardiac hypertrophy or dilatation.

Pulsation at the Right Sternal Border.—Systolic pulsation in the first and second intercostal spaces adjacent to the right sternal border accompanies aneurysm of the ascending aorta. Systolic pulsation in the second, third and fourth interspaces occurs when the heart is displaced toward the right by the pressure of fluid or gas in the left pleural sac, or by the traction of adhesions between the pericardium and the right pleura.

Presystolic pulsation in the same area occurs in the presence of right auricular dilatation, and occasionally when the heart is exposed to the thoracic wall as a result of fibroid retraction of the anterior border of the right lung.

Pulsation at the Left Sternal Border.—Systolic pulsation adjacent to the left sternal border in the second and third interspaces, most pronounced between the left sternal and parasternal lines, accompanies aneurysm of the descending portion of the aortic arch. Fibroid retraction of the anterior border of the left lung or displacement of the heart toward the left causes the systolic pulsation of the conus arteriosus to become visible in the second, third and fourth interspaces adjacent to the sternum.

Presystolic pulsation in the second left interspace adjacent to the sternum points to dilatation of the left auricle as the causative lesion.

Sternal Pulsation.—Systolic pulsation in the superior sternal region, frequently attended by coincident episternal pulsation, is indicative of erosion of the sternum by aneurysmal dilatation of the aorta, or less frequently aneurysm of the innominate artery. The pulsation is accompanied by boring pain over the site of the erosion.

Episternal Pulsation.—The student of physical diagnosis is early impressed by the great frequency with which pulsations are encountered in the episternal notch. Frequently it is not easy to assign the pulsation to its proper cause, as pulsation in this restricted space is encountered in a variety of conditions.

Episternal pulsation is a normal phenomenon in many elderly subjects; it occurs as a sign of essential or secondary anemia; it follows excessive physical effort or the ingestion of stimulants; and it is prone to develop during the course of cardiac neuroses. The pulsation may be caused by the presence of a cervical rib, or it may be due to an abnormal course of the great vessels at the root of the neck. A similar systolic pulsation is encountered in this region when the subclavian artery is exposed to the thoracic wall by fibroid retraction of the pulmonary border.

But it should be borne in mind that systolic pulsation of the episternal notch attends simple dilatation and aneurysm of the transverse portion of the aortic arch with great constancy.

Supraclavicular Pulsation.—Pulsation in the supraclavicular region may be of *arterial* or *venous* origin. Systolic pulsation in the right supraclavicular region is noted in connection with aneurysm of the innominate or subclavian artery, and the pulsation is usually accompanied by a palpable thrill. In the presence of left ventricular hypertrophy, with or without regurgitation at the aortic orifice, there is frequently bilateral *carotid pulsation* in the supraclavicular region. In this instance it can be demonstrated that the systolic pulsation occurs over the course of the carotid artery, along a line drawn from the sternoclavicular articulation to a point equidistant between the angle of the jaw and the mastoid process. Carotid pulsation is likewise encountered in the presence of aneurysm of the aortic arch, arteriosclerosis, exophthalmic goiter, anemia, and after the ingestion of stimulants.

Presystolic **jugular pulsation**, representing the physiological *negative venous pulse,* is occasionally to be detected in the spare subject. The pulsation is generated by the sudden arrest of the venous column during auricular systole; and the pulsation is timed by palpation of the jugular vein with the finger-tips of the left hand while applying the tips of the fingers of the opposite hand to the opposite carotid artery or to the cardiac impulse.

Systolic pulsation of the jugular vein, representing the pathological *positive venous pulse,* caused by direct regurgitation of blood into the right auricle from the right ventricle during systole, is significant of tricuspid regurgitation. While the provocative lesion of the positive venous pulse is in the great majority of cases tricuspid regurgitation, such a pulse is also produced occasionally by mitral regurgitation associated with a patent foramen ovale. The positive venous pulse is frequently accompanied by systolic pulsation of the liver.

Turgescence of the jugular veins is noted in the presence of compression of the large veins by mediastinal tumor, aneurysm, enlarged mediastinal glands, or adhesive mediastinitis. If the turgescence is bilateral, involving both jugular veins, the underlying cause is cardiac insufficiency or compression of the superior vena cava, the innominate or the jugular veins; whereas, unilateral turgescence is noted in the presence of compression of one innominate or jugular vein.

Normally exhibiting moderate distention during expiration, this expiratory overfullness of the jugulars amounts to engorgement or turgescence in subjects of bronchial asthma and hypertrophic emphysema. A reversal of this expiratory distention of the jugulars, in which inspiratory turgescence is encountered, constitutes *Kussmaul's sign* of chronic adhesive pericarditis.

Diastolic collapse of the jugular veins accompanies chronic adhesive pericarditis, constituting *Friedreich's sign* of this disease. In chronic adhesive pericarditis the traction of pleuropericardial adhesions produces retraction of the chest wall during ventricular systole. During the succeeding diastole the elastic walls of the thorax expand, and thus aspirate the blood from the great veins at the base of the heart, resulting in diastolic collapse of the jugular veins.

Unilateral jugular collapse, which does not disappear when the vein is compressed immediately above the clavicle, is indicative of lateral sinus thrombosis.

Hepatic Pulsation.—Systolic pulsation of the liver accompanies tricuspid regurgitation and is apt to be attended by signs of general venous stasis, as edema of the extremities, ascites, and cyanosis. Not infrequently a hepatic pulsation which is not visible is yet distinctly palpable upon bimanual palpation of the hepatic region. True expansile pulsation of the liver, which occurs with incompetence of the tricuspid valve, must be differentiated from the impulse which is frequently transmitted to the liver by the impact of an overacting right ventricle.

Epigastric Pulsation.—Systolic pulsation of the epigastrium is occasionally noted in the normal subject without possessing untoward significance. Under these circumstances it is usually to be attributed to physiological bathycardia or to shortness of the sternum. Systolic epigastric pulsation likewise accompanies hypertrophy of the right ventricle, and is also noted when the heart is displaced toward the right so that the apex lies behind the sternum.

Diastolic pulsation of the epigastrium accompanies anemia and neurasthenia, and is noted in subjects of chronic gastric indigestion with great frequency. Diastolic pulsation in this region likewise accompanies a tumor of an abdominal organ overlying the aorta, the pulsation being transmitted to the tumor during each pulsation of the artery. A similar pulsation, which is readily palpable if not visible, attends aneurysm of the abdominal aorta.

Epigastric pulsation arising from the abdominal aorta occupies a position in the lower epigastrium and is situated slightly to to the left of the median line, whereas the epigastric pulsation of right ventricular hypertrophy occupies the superior portion of the epigastrium in the costal angle.

THORACIC RETRACTION (BROADBENT'S SIGN)

In chronic adhesive pericarditis, in the presence of extensive adhesions between the pericardium and the diaphragm, with each ventricular systole there is noted a systolic retraction of the thoracic wall. Broadbent directed attention to the frequency with which systolic retraction is visible upon the left side of the thorax posteriorly, below the angle of the scapula in the tenth and eleventh interspaces in this disease. There is frequently to be noted a similar systolic retraction of the anterior chest wall in the eighth and ninth interspaces in the left parasternal line.

As has been noted, aside from chronic adhesive pericarditis, localized systolic recession of the thoracic wall in the vicinity of the cardiac impulse is noted in the presence of excessive hypertrophy of the left ventricle.

TRACHEAL TUG (OLIVER'S SIGN)

When adhesions have become established between an aortic aneurysm and the trachea or left bronchus, an appreciable descent of the trachea occurs with each pulsation of the aneurysmal sac. In eliciting Oliver's sign the examiner should stand behind the patient, who occupies the sitting posture with the lips closed and with the chin elevated in order to exert moderate traction upon the trachea through the cervical fascia. In this position the examiner should insert the tips of the thumb and index finger under the lower edge of the cricoid cartilage and exert gentle upward pressure upon these structures. In the presence of aneurysm of the aorta a systolic tug will be appreciated by the palpating digits.

In drawing conclusions from tracheal tugging, however, the examiner should bear in mind that while the sign is present with great constancy with aneurysm of the aortic arch, it has also been elicited in the presence of simple dilatation of the aorta; and in the presence of excessive hypertrophy of the heart a similar movement has been imparted to the trachea by strong pulsations in the innominate and carotid arteries.

VALVE SHOCK

Valve shock is due to the impact of closure of the valves of the heart, and it can be appreciated when the palm of the hand is applied flatly over the valve area in question. The shock is occasionally to be elicited over the areas of the auriculoventricular valves, but not with the same constancy as is the case with the aortic and pulmonary valves. In either case the thickness of the thoracic wall, the state of the superimposed pulmonary tissues, and the state of the myocardium and arteries exert a striking influence upon the intensity of the shock as appreciated by the palpating hand.

Valve shock elicited over the auriculoventricular valves is systolic in time, corresponding to the closure of the cusps of these valves; whereas the shock over the aortic and pulmonary valves occurs during the first portion of ventricular diastole, at the instant of closure of the cusps of the semilunar valves.

Accentuation of the shock over the pulmonary area is noted in the presence of right ventricular hypertrophy, and when the pressure in the pulmonary circulation is raised as a result of valvular lesions of the left side of the heart or obstructive diseases of the lungs.

The aortic shock is accentuated in the presence of left ventricular hypertrophy, and in arteriosclerosis and chronic renal disease, conditions which are attended by hypertension of the greater circulation.

THRILLS

Upon light palpation of the precordia of a patient who is the subject of valvular disease of the heart, aneurysm, or anemia, a local vibration of the thoracic wall, or *thrill*, is apt to be detected at one or more points. The sensation which is appreciated during palpation is somewhat similar to that which is experienced upon placing the hand upon the throat of a purring cat, hence the term *frémissement cataire,* occasionally employed to describe it.

Thrills are endocardiac, vascular or hemic in origin; and point of time they are presystolic, systolic or diastolic, as t occur prior to, during, or after ventricular systole.

A thrill of endocardiac origin is generated by the same me anism which is responsible for the production of an endocard murmur; namely, a narrowing of an orifice through which blood stream is propelled into a larger chamber of the heart. long as the normal quota of blood of normal density flows throu normal orifices, no abnormal vibration is generated; but when lumen at one point is diminished, the blood which passes throu

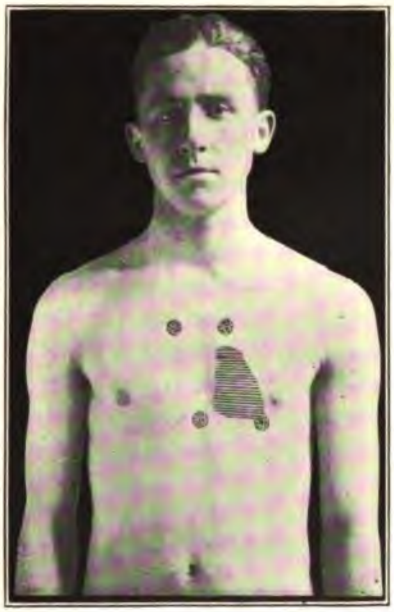


Fig. 122-*A*.—Sites of palpable thrills and pericardial friction fremitus.

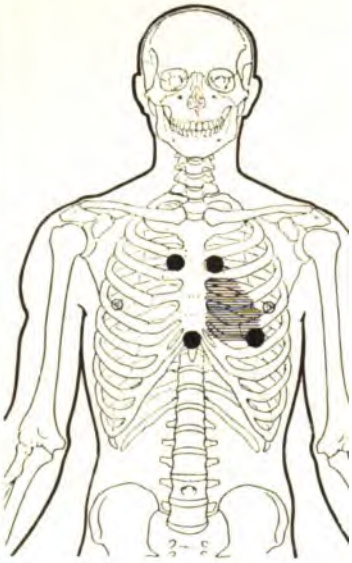


Fig. 122-*B*.—Sites of palpable thrills pericardial friction fremitus.

this stenotic orifice into the wider chamber beyond is thro into eddies, creating the so-called "fluid veins," the vibrati of which are conveyed to the precordia as a tactile vibration, thrill, which is the tactile equivalent of a murmur.

As thrills of endocardiac origin are confined to limited ar of the precordia, they are most surely detected in the first stance by light palpation with the palm of the hand, after wh they may be more definitely defined by finger-tip palpation. Fi pressure with the palm frequently suffices to obliterate a thril

The intensity of a thrill is very variable, as the vibratio very prone to disappear in the case of a feebly acting heart, and to reappear with improvement in the state of the myocardium. The quality of a thrill varies with the rate of the vibrations which call the thrill into being, rapid vibrations of the blood stream producing fine thrills and murmurs of high pitch, whereas less rapid vibrations result in coarse thrills and murmurs of lower pitch.

In general, a systolic thrill at the base of the heart is indicative of aortic aneurysm, exophthalmic goiter, or structural changes in the aortic or pulmonary valves. The thrill of aortic aneurysm is commonly localized in the second and third interspaces to the right of the sternum; that of aortic stenosis is detected in the second right intercostal space adjacent to the sternal border; while the thrills of pulmonary stenosis and exophthalmic goiter are palpable in the second left interspace adjacent to the border of the sternum.

Diastolic thrills at the aortic and pulmonary areas occasionally accompany regurgitant lesions of the aortic and pulmonary valves; but thrills are demonstrable with greater constancy in the presence of stenotic than of regurgitant lesions.

A presystolic thrill localized over the cardiac apex in the fifth left intercostal space is a very good sign of mitral stenosis. However, a similar thrill has been found to accompany the Flint murmur of aortic regurgitation in this area. Only occasionally is a systolic thrill to be detected over the apex in the course of mitral regurgitation.

A presystolic thrill is occasionally to be elicited over the tricuspid area in the presence of tricuspid stenosis; while a systolic thrill in the same area is occasionally to be detected in tricuspid regurgitation.

In the interpretation of precordial vibrations the examiner should remember that in the presence of moderate cardiac hypertrophy in a subject with a thin chest wall, or in the presence of fibroid retraction of the anterior border of the left lung in the anemic subject, the impact of the conus arteriosus of the right ventricle against the thoracic wall may generate a physiological thrill in the absence of valvular disease.

PERICARDIAL FRICTION FREMITUS

Friction fremitus of pericardial origin is generated during the evolution and course of acute fibrinous pericarditis or during pericarditis with effusion. In the latter disease the fremitus prone to disappear with the gradual accumulation of the ef sion, which separates the layers of the pericardium, though is not uncommon for the fremitus to persist at the base of heart during the height of the effusion.

Pericardial friction fremitus is usually to be detected in second, third and fourth interspaces along the left border of sternum; it does not extend beyond the limits of the precord it is frequently attended by moderate pain; and the pain accentuated by pressure exerted over the lower portion of sternum.

Pleuropericardial friction fremitus is elicited over the tribution of the lappet of lung which is in apposition with right ventricle, or over the portion of the pericardium which in intimate relation with the anterior thoracic wall in the incisu cardiaca. In the first case the fremitus is abolished with recession of the anterior pulmonary border during forced expi tion, whereas in the second instance the fremitus is enfeebled abolished during forced inspiration, when the anterior pulm ary border intervenes between the pericardium and the cos pleura.

THE PULSE

By the term *pulse* is understood the expansion and subsequ retraction of an artery following each systole of the left ventri Usually the radial artery at the wrist is the site selected studying the pulse on account of its readiness of access; but ot arteries, as the temporal, carotid, or femoral may likewise utilized in the analysis of the pulse. The pulse should be stud by digital examination, and by instrumental means with sphygmograph or polygraph.

PALPATION OF THE PULSE

During palpation of the radial pulse the patient should occu the sitting or recumbent posture, with the arm extended upon level with the heart and supported by the left hand of the aminer, who, seated beside the patient, should first apply the t of the first three fingers of the right hand over the course of radial artery. The examiner should then pass the finger-t longitudinally along the course of the artery and transvers across the artery, and, while rolling the vessel beneath fingers, study the several factors which enter into the analysis of the pulse. When the radial artery is so palpated it is observed that at each pulse wave the artery is changed from a flat tube into a circular one, and moreover, that the vessel lengthens or straightens appreciably during this period.

It is desirable in all cases to count the pulse for a full minute in recording the rate of the pulse. The pulse may, however, be counted during twenty seconds and the result multiplied by three; or it may be counted during thirty seconds and the result multiplied by two. When the pulse is so rapid that it is impossible to count all of the individual beats, an approximate estimate of the frequency of the pulse may be made by endeavoring to count every other beat, or the examiner may make dots with a pencil held in the unengaged hand and count the number of dots which are made during a minute. When it is suspected that each systole of the ventricle does not produce a radial pulsation, the examiner may arrive at a correct conclusion by auscultation of the area of the cardiac impulse and by counting the systoles simultaneously with the palpation of the radial pulse.

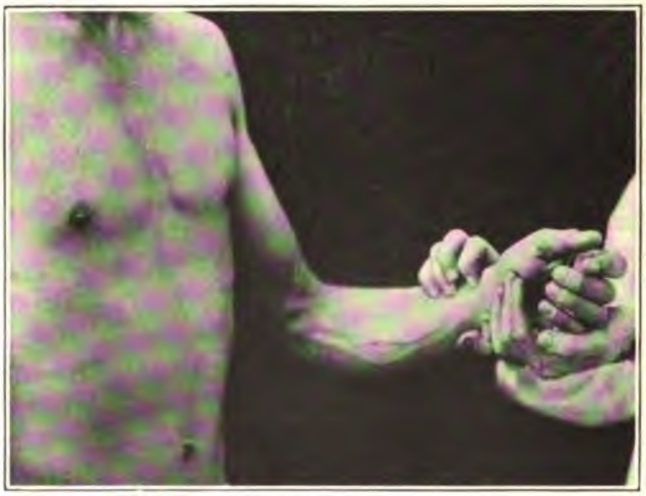


Fig. 125.—Palpation of the radial pulse.

SPHYGMOGRAPHY

The several elements which make up the radial pulse are graphically reproduced by the sphygmograph, the instrument originally devised by Marey and improved by Dudgeon, or by t sphygmocardiograph of Jaquet or MacKenzie.

In the practice of sphygmography the instrument is adjusted the wrist in such manner that the diaphragm of the instrume overlies the radial artery, the exact course of which has be marked upon the integument with a dermographic pencil. Wh the proper adjustment is obtained the pulsation of the artery communicated to the writing needle, which records the sphygm gram upon a strip of smoked paper, which is driven through t instrument by clock work. As the Dudgeon instrument is timed that a five inch slip of paper passes through it in t seconds, the pulse rate per minute is readily calculated.

The normal sphygmogram consists of an upstroke or *anacrot limb*, an *apex*, a down-stroke or *catacrotic limb*, and a *base lin*

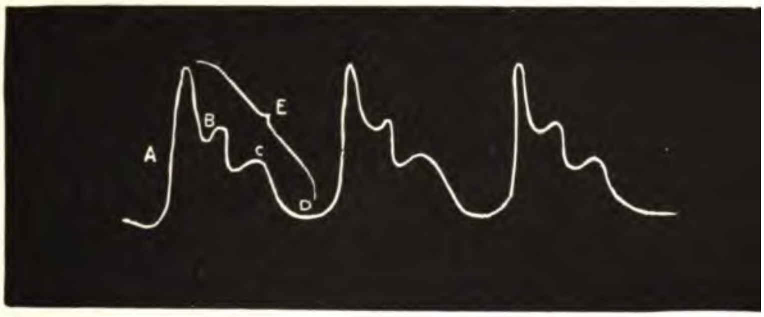


Fig. 126.—Normal sphygmogram. *A*, Anacrotic limb; *E*, Catacrotic limb; *B*, Tid wave; *C*, Dicrotic wave.

which represents the level of the lowest points of the sever strokes.

The anacrotic limb of the tracing corresponds with the eje tion of blood into the arterial system during ventricular systol When the anacrotic limb has attained a variable height, t catacrotic limb descends gradually to the base line, the juncti of the two limbs forming the angle or apex of the sphygmogra

As the catacrotic limb descends to the base line it is interrupt by two distinct waves. The first wave, occurring early duri the descent, the *tidal* or *predicrotic wave*, is due to the seconda contraction and expansion of the artery following its distenti during ventricular systole. The second wave, the *recoil* or *dicrot wave*, is to be attributed to the recoil of the column of circulati blood, whose course toward the ventricle is interrupted by closu of the aortic valve. There are occasionally to be noted, in add tion to these two principal waves, minute oscillations of the catacrotic limb due to the normal elasticity of the arterial wall.

VARIATIONS IN THE SPHYGMOGRAM

In the interpretation of sphygmographic tracings in clinical work, attention should first be directed to the course pursued by the base line. An irregular base line is encountered in the presence of arterial hypotension attended by dyspnea. Under these circumstances the base line presents an elevation during expiration, and it falls during inspiration.

Irregularity in the spacing of the limbs of the tracing is noted in the presence of cardiac arrhythmia involving the temporal sequence of the ventricular contractions. Variations in the height of the strokes are likewise encountered in cardiac arrhythmia involving the force of the consecutive ventricular systoles. An abnormally long anacrotic limb is noted in the presence of ventricular systole, of considerable power, but frequently of brief duration, as in aortic regurgitation and in arterial hypotension. An abnormally short anacrotic limb is indicative of the expulsion of a diminished quantity of blood from the ventricle during systole, occurring as a result of mitral regurgitation, aortic stenosis, or increased peripheral resistance.

The direction pursued by the anacrotic limb should be studied in every tracing. A vertical anacrotic limb, with a very acute angle or apex, is significant of ventricular systole of brief duration and low arterial tension, a state which obtains particularly in aortic regurgitation. An oblique course of the anacrotic limb, on the contrary, is suggestive of tardy emptying of the left ventricle as a result of aortic stenosis, mitral regurgitation, or increased peripheral resistance incident to transient angiospasm, arteriosclerosis, or chronic renal disease.

The angle which is formed by the junction of the anacrotic and catacrotic limbs, representing the apex of the sphygmogram, possesses potent diagnostic and prognostic significance. A sharp apex is indicative of arterial hypotension, occurring as the result of anemia, transient vasomotor relaxation, or aortic regurgitation. A broad apex, on the contrary, indicates powerful ventricular systole in the presence of an obstacle to the onward course of the blood stream, such as occurs in arteriosclerosis or chronic renal disease; or it is due to prolongation of ventricular systole in the presence of aortic stenosis with left ventricula hypertrophy.

Accentuation of the *tidal wave* of the sphygmogram is noted i conditions associated with arterial hypertension, notably arteriosclerosis and chronic renal inflammation; whereas, a diminutiv tidal wave is encountered in conditions of cardiac asthenia, during transient vasomotor paresis, and in the presence of regurgitant lesions at the aortic and mitral valves.

Accentuation of the *dicrotic wave* is indicative of low arteria tension combined with ventricular contractions of moderat strength. With less constancy a marked dicrotic wave is observe in the presence of arterial hypertension when ventricular failur is imminent. Diminution in the dicrotic wave, the wave coincidentally occupying a plane high above the base line, occurs wit arterial hypertension with adequate compensation by the lef ventricle, notably in the presence of arteriosclerosis and chroni nephritis.

SPHYGMOCARDIOGRAPHY

With the sphygmocardiograph of Jaquet or MacKenzie th examiner is enabled to record synchronous tracings of the radia pulse, the cardiac impulse, and a third pulsating area, whether i be the carotid artery, jugular vein, hepatic pulsation, or pulsatio in other sites adjacent to the precordia.

In the practice of sphygmocardiography, after adjusting th instrument to the wrist, the second receiver is strapped in position over the site of the cardiac impulse, and the third receive is held in apposition with the integument over the course of th jugular vein when it is desired to record the impulses generate in these respective portions of the circulatory apparatus. Wit the three tracings thus obtained the examiner may compare th temporal incidence and any pathological variations in the severa portions of the sphygmocardiogram.

The *sphygmogram* obtained with these instruments presents th same elements as have been described under sphygmography, bu the tidal and dicrotic waves are recorded more clearly than the are with the Dudgeon sphygmograph.

The normal *cardiogram* thus recorded presents a perpendicula anacrotic limb, corresponding to the beginning of ventricula systole, followed by a very obtuse angle or broad apex, the "systolic plateau," which corresponds with the impact of the ventricle against the chest wall during systole. From the apex th catacrotic limb falls slowly to the base line, and is followed immediately by a minor elevation, corresponding to the earliest portion of ventricular systole. Occasionally the anacrotic limb of the cardiogram is preceded by a minor elevation corresponding to auricular systole, but this wave is not frequently demonstrable in the normal cardiogram.

The *venous sphygmogram,* recorded over the jugular vein, representing the physiological or *negative venous pulse,* consists of three waves, which are dependent upon movements of the right side of the heart for their development.

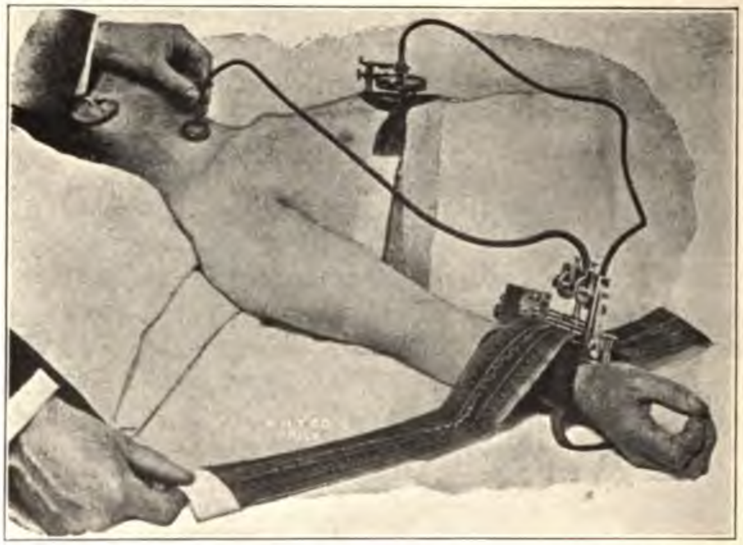


Fig. 127.—Jaquet sphygmocardiograph. Simultaneous tracings of radial pulse, cardiac impulse, and carotid pulse.

The first wave, the *auricular* or *a-wave,* corresponds to auricular systole, and is presystolic in time. It is produced by the sudden arrest of the onward flow of the venous column during auricular systole.

The second wave, the *systolic* or *s-wave,* coincides with the beginning of ventricular systole, occurring at the moment of projection of the tricuspid valve segments into the chamber of the right auricle, producing in this wise a reversed wave in the veins. As the third wave of the jugular sphygmogram is also systolic in time, the s-wave is frequently termed the *protosystolic wave.*

The third wave, the *ventricular* or *v-wave,* accompanies the latter portion of ventricular systole. This wave presents a double curve, which is to be attributed to the projection of the tricuspid cusps into the chamber of the auricle and to the upward displacement of the tricuspid valve during the latter portion of ventricular systole.

The notch which is noted between the a-wave and the s-wave corresponds to the commencement of auricular relaxation, while the notch between the s-wave and the v-wave coincides with auricular diastole. Similarly, the notch which follows the v-wave corresponds to the diastole of the ventricle.

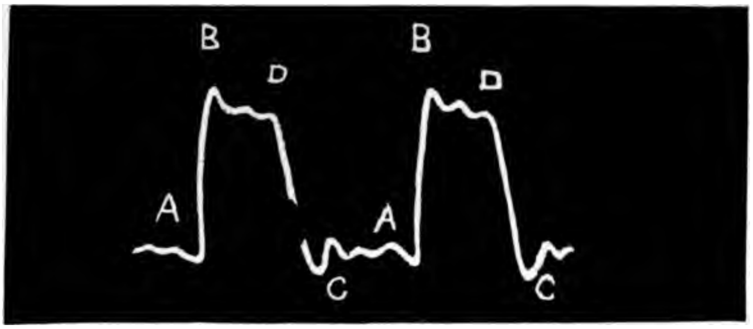


Fig. 128.—Normal cardiogram. *A-B*, Anacrotic limb; *B-C*, Catacrotic limb; *B-D*, systolic plateau.

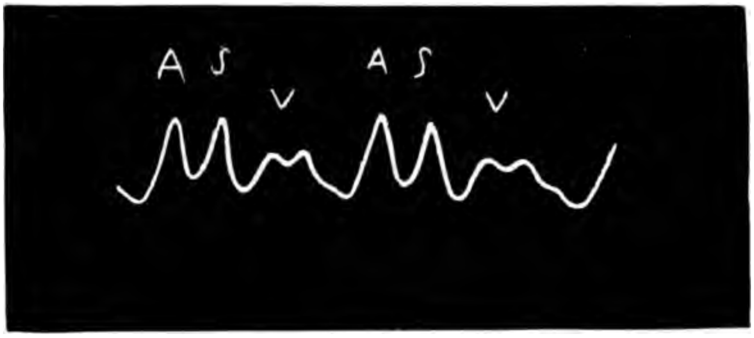


Fig. 129.—Normal jugular sphygmogram. *A*, Auricular wave; *S*, Systolic wave; *V*, Ventricular wave.

VARIATIONS IN THE PHLEBOGRAM

The magnitude of the notch which intervenes between the a-wave and the s-wave represents graphically the interval of time which is required for the conduction impulse to pass from the auricles to the ventricles; hence, augmentation of this notch points to a lesion in the conduction bundle of His.

In the presence of increased venous pressure with engorgement of the right ventricle, the v-wave occurs appreciably earlier in the phlebogram, rises to a greater height than normally, and is moderately prolonged.

With the establishment of tricuspid regurgitation the s-wave of the jugular sphygmogram is replaced by a large v-wave, representing the pathological *positive venous pulse.*

ANALYSIS OF THE PULSE

In analyzing the radial pulse the examiner should study the points enumerated, variations in one or several of which may be encountered.

1. Condition of the arterial wall.
2. Size of the artery.
3. Rate.
4. Rhythm.
5. Tension.
6. Volume.
7. Force.
8. Duration.
9. Bilateral symmetry of the pulses.

Condition of the Arterial Wall.—In studying the condition of the wall of the radial artery the examiner should endeavor to exclude the blood from the artery by compression with the index and third fingers, in the meantime rolling the artery beneath the middle finger and the subjacent radius. During this maneuver counterpressure should be applied to the dorsum of the wrist with the thumb. The wall of the normal radial artery is soft and yielding, and is to be distinguished with difficulty from the surrounding tissues. Any departure from the normal elastic state of the artery is of diagnostic significance; but in this connection the examiner should not mistake increased tension of the pulse for thickening of the arterial wall.

In the aged subject and in the younger subject in the presence of arteriosclerosis due to alcoholism, syphilis, gout, or other cause, the arterial wall presents varying grades of resistance to manual compression. In advanced stages of arterial fibrosis the artery may frequently be rolled between the fingers and the lower end of the radius, yielding a tactile impression similar palpation of a hard reed or pipe stem. It is to be borne in min however, that the detection of sclerosis of the radial artery do not necessarily signify a similar grade of hardening of the aor and coronary arteries. In other instances the artery is bead and more or less tortuous, owing to the presence of atheromato plaques or beads in the arterial wall.

The Size of the Artery.—The radial artery may be larger smaller than normal as a result of congenital deformity, witho possessing untoward significance. Transient changes in the si of the arterial pulsation, dependent upon changes in the myoca dium and disturbances in the vasomotor system, are noted in t section dealing with the volume of the pulse.

Rate.—The normal pulse rate in the adult subject varies fro 70 to 80 beats per minute. The rate of the pulse, however, modified by physiological factors, such as the age and sex of t subject, the size of the body, the position assumed by t patient, and the relation of the time of recording the pulse the ingestion of food.

At birth the pulse rate is approximately 130 to 140 beats p minute; during the first year of life it is 115 to 130; at t seventh year it averages 85 to 90; while in the aged subject drops to 60 to 70 beats per minute. In women the pulse is usual moderately increased in frequency over the rate obtaining in t adult male subject.

The size of the body exerts a slight influence upon the rate the pulse, the rate being lower in large subjects than it is persons of small stature. The position of the patient influen the pulse rate, the pulse being constantly more rapid wh counted in the upright posture than when it is taken with t patient in the recumbent position. The rate is likewise quicken for an hour or two following a full meal. Exercise and mental emotional excitement serve to cause a transient increase in t pulse rate, not infrequently doubling the rate for the individu Finally, the ingestion of excessive quantities of tea or coff or the excessive use of tobacco may be responsible for a transi elevation of the pulse rate.

Pathologically, elevation of the pulse rate, evinced by t *pulsus frequens,* is noted in disturbances of the innervation the heart, stimulation of the sympathetic innervation from th acic or abdominal disease and inhibition of the vagus innervati resulting in reflex tachycardia. Increase in the pulse frequen and in intoxication from alcohol, opium or digitalis the p rate is frequently slowed to a remarkable degree.

Diminution of the pulse rate may be due to irritation of trunk of the vagus nerve by mediastinal tumor, enlarged me tinal glands, aortic aneurysm or extensive empyema. Simil depression of the sympathetic innervation, with consequent bition of the accelerator impulses to the myocardium, resul slowing of the pulse.

In the presence of arterial hypertension arising from art sclerosis or chronic renal inflammation, the slow and vigo systole of the hypertrophied left ventricle is attended by mo ate slowing of the radial pulse.

Rhythm.—In the normal subject the several pulse beats ar uniform force, and the beats are separated by uniform i vals. It follows, therefore, that a disturbance in the rhyth the pulse may have reference either to the force or to the poral sequence of the beats.

Arrhythmia of the pulse is betrayed by a deviation from normal orderly sequence of the beats without the omissio beats. It is to be observed during the course of acute fever the presence of valvular lesions of the heart, during diges disturbances, following the excessive use of tobacco, in les of the brain, in gout, and in the presence of myocardial dege ation, in which, indeed, it may be the only sign of the dis Irregularity of the pulse is not infrequently noted in elderly jects, however, without possessing untoward significance.

Intermission is the occasional omission of a pulse beat. omission may occur at irregular intervals, or the omission follow an orderly sequence, every third or fourth beat b omitted. Intermission may be a transient phenomenon, o may persist throughout life without possessing untoward sig cance. It is usually to be attributed to nervous exhaustion o the abuse of tobacco.

In the analysis of a case of intermission it is important to termine by auscultation of the precordia whether the omis of the radial pulse is due to an omission of ventricular sys *pulsus deficiens;* or whether it is due to a ventricular contrac which is too feeble to produce a radial pulse, the *pulsus i mittens.*

When the omissions of the pulse beats follow a regular seque when they are rhythmically irregular, the result is the produc of an *allorrhythmic* pulse. In this variety of intermission bel with the glottis closed, or by holding the breath after full inspiration.

The paradoxical pulse is noted during the extreme inspiratory dyspnea attending laryngismus stridulus or spasmodic croup. It has been noted in the presence of pericarditis with effusion, chronic adhesive pericarditis, mediastinal inflammatory disease and tumor, and during extreme cardiac asthenia.

The antithesis of the paradoxical pulse is occasionally observed, in which the radial pulse trails off and becomes imperceptible during expiration (Riegel's pulse).

Irregularities in the force of the successive pulse beats are encountered in the *pulsus alternans,* with or without disturbance in the temporal relations of the individual beats. This type of arrhythmia is of grave prognostic significance, pointing to exhaustion of the myocardium during the course of arteriosclerosis, chronic nephritis, fibrous myocarditis, or angina pectoris.

Volume.—The volume or size of the pulse is dependent upon the size of the artery, the amount of blood which is expelled during ventricular systole, the ability of the aortic valve to prevent regurgitation of the blood column, and upon the state of the vasomotor nervous system. Thus, in the presence of powerful ventricular contractions associated with vasomotor depression during the course of acute febrile diseases the radial pulse is full and bounding, constituting the *pulsus magnus.* Similarly, hypertrophy of the left ventricle with competence of the aortic valve is attended by the pulsus magnus.

When the artery is palpably filled with blood in the intervals between the individual pulse beats, indicating the presence of a large bulk of circulating blood, the pulse is termed the *pulsus plenus.*

In the presence of cardiac asthenia, or when as a result of regurgitant or stenotic lesions at the aortic or mitral valves a diminished quota of blood is expelled from the ventricle during systole, the radial pulse is of small volume, the *pulsus parvus.* A similar pulse is noted in the presence of extreme grades of anemia as a result of diminution in the total bulk of the circulating blood.

When the artery is palpably empty and readily compressible during the intervals between the pulse beats, indicating a diminution in the bulk of the circulating blood, the pulse is termed the *pulsus vacuus.*

Force.—The force of the pulse depends upon the energy with which the left ventricle contracts and upon the elasticity of the rterial walls. If the ventricle is hypertrophied but the arterial valls have lost their elasticity as a result of fibrosis, much of the orce of the heart consumed in the expulsion of the blood is lost hrough the absence of the elastic recoil of the arteries.

In general, the force of the pulse is increased in conditions atended by cardiac hypertrophy, and it is diminished in the presnce of cardiac asthenia and dilatation. Moderate stimulation of he vagus nerves increases the pulse force by slowing the heart nd increasing its period of repose; but if the stimulation is exreme, the ventricular contractions are so few that the diminished lood content of the arterial system is not sufficient for the powerul ventricular systole to act upon, and the force of the pulse is onsequently diminished.

Tension.—The tension of the pulse is influenced by the power nd the rate of ventricular contraction, the volume of the circulatng blood, the elasticity of the arterial walls, and the degree of eripheral resistance to the blood flow. In the presence of arterioclerosis and chronic renal disease associated with hypertrophy of he left ventricle, arterial tension is raised (hypertension), as vinced by a hard pulse, which is compressed with distinct effort, he *pulsus durus.* Transient angiospasm is attended by a similar ulse of high tension, but the alteration in tension in this instance s subject to fluctuations and is not permanent.

When, on the other hand, the output of blood from the ventricle s diminished by cardiac dilatation or valvular disease, combined vith vasodilatation or a diminution in the amount of circulating lood as a result of hemorrhage, anemia or cachexia, the arterial ressure is diminished (hypotension), and the pulse is soft and eadily compressible, the *pulsus mollis.*

Occasionally during the course of a continuous fever associated vith vasodilatation, there is noted a pulse of low tension and dininished rate but of full volume, in which there is a reduplication ppreciable to the palpating fingers as a minor beat, following the rincipal pulse beat, the *dicrotic pulse.* The dicrotic palpable vave is to be attributed to excessive elasticitiy of the arteries comined with general relaxation of the smaller arterioles. The pulse s occasionally demonstrable in the presence of imperfectly comensated mitral regurgitation, during typhoid fever, and in states f anemia and exhaustion.

Very rarely in extreme grades of aortic stenosis there is a palable tidal wave of the artery, following immediately upon the rincipal beat, corresponding to the tidal wave of the sphygmothe presence of arterial hypertension, and the second wave is appreciable immediately subsequent to the principal pulse beat.

Duration.—The duration of the pulse is determined by the duration of ventricular systole, the elasticity of the arterial walls, and the degree of peripheral resistance.

The duration of the pulse is increased in the slow, sluggish *pulsus tardus,* which is always associated with increased peripheral resistance due to constriction of the small arterioles, such as occurs in arteriosclerosis, chronic renal disease, and angina pectoris. The sphygmogram of this pulse shows a gradual up-stroke, a well sustained apex or plateau, and a gradual fall to the base line. The pulse is likewise observed in aortic stenosis, in which case it is to be ascribed to the prolonged systole of the left ventricle.

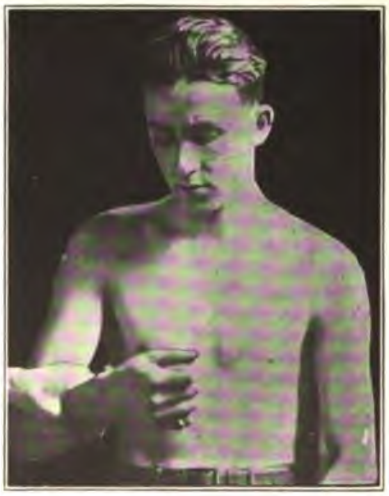


Fig. 132.—Method of detection of water-hammer pulse.

The duration of the pulse is diminished with the production of a very brief pulse wave, the *pulsus celer,* in conditions associated with diminished peripheral resistance as a result of relaxation of the arterioles in the course of acute fevers. The pulse is also encountered in the presence of aortic regurgitation, constituting the *water-hammer* or *Corrigan pulse* of this disease. The pulse is characterized by a sudden and full expansion of the artery, followed by a sudden collapse of the vessel under the fingers. The pulse is readily demonstrated by grasping the wrist and elevating the arm above the level of the heart. The sphygmogram of the pulsus celer shows an abrupt up-stroke, followed by an acute angle and by a rapid fall of the down

Bilateral Symmetry of the Pulses.—In the normal subject there is no appreciable difference in the radial pulse as recorded at the two wrists. This symmetry of the pulses may, however, be disturbed even to the total absence of the pulse at one wrist. Aneurysm of the ascending aorta or innominate artery may retard the right radial pulse; while aneurysm of the subclavian, axillary or brachial artery may cause retardation upon either side of the body.

Fracture of the bones of the arm or injuries producing cicatricial compression of the artery in the axilla or arm, as well as compression by tumors or enlarged glands, will alter the character of the pulse at the wrist and cause asymmetry of the pulses.

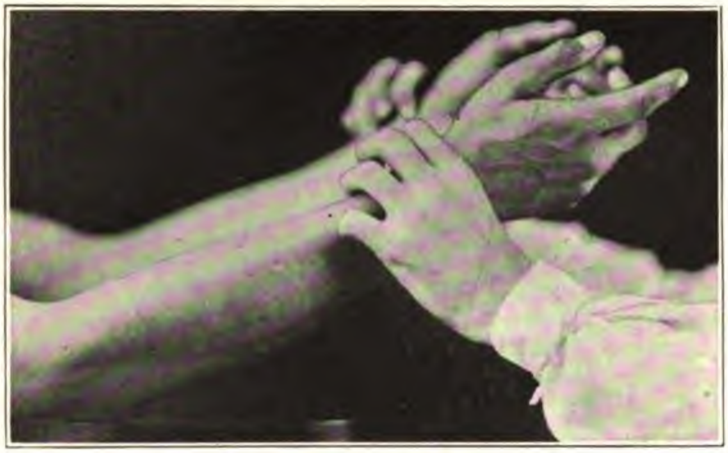


Fig. 133.—Testing the symmetry of the radial pulses.

Pneumothorax or massive pleural effusion, by compression of the subclavian artery, may cause a retardation or may alter the character of one radial pulse.

THE CAPILLARY PULSE

Systolic pulsation in the capillaries is sometimes a normal phenomenon; it may be the result of temporary loss of vasomotor tone during anemia or febrile diseases; but it is also a very valuable sign of aortic regurgitation or Corrigan's disease.

There are several methods of demonstrating capillary pulsation. A serviceable method is by blanching the finger nail by the exertion of moderate pressure upon the tip of the nail, whereupon a systolic flushing and a diastolic blanching of the subungual tissues will be observed. Another method of demonstrating the phenomenon is by drawing the nail over the forehead and producing a line, which is alternately flushed and blanched during ventricular systole and diastole. A third method of detecting capillary pulsation is by covering the lower lip by a glass slide and observing the systolic flushing and diastolic blanching of the lip which is compressed by the slide.

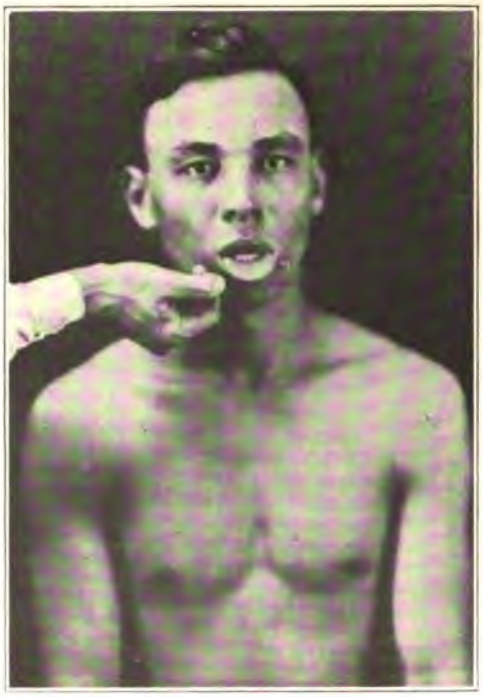


Fig. 134.—Demonstration of capillary pulse.

The capillary pulse is frequently accompanied by visible pulsation in the carotid arteries, and by pulsation in the veins of the dorsum of the hand or foot.

THE CENTRIPETAL VENOUS PULSE

A visible pulsation, the centripetal or penetrating venous pulse, is occasionally visible in the veins of the dorsum of the hand or foot, or in the delicate mammary veins. This pulsation is most frequently associated with aortic regurgitation, and less commonly with anemia, constituting in the first instance an exaggeration of the capillary pulse of Quincke.

CHAPTER XIV

PERCUSSION

Percussion is employed in the study of cardiovascular disea
principally in the determination of the position of the heart,
the detection of alterations in its shape and size, and in t
detection of the presence of fluid in the pericardial sac. Pe
cussion is likewise employed to advantage in estimating t
dimensions of the area of dullness produced by the great vess
springing from the base of the heart in the area of vascul
dullness.

The size, shape and position of the heart within the thora
cavity are determined by outlining upon the surface of t
thorax the areas of relative and absolute cardiac dullness.

AREAS OF CARDIAC DULLNESS

When the examiner percusses toward the heart from vario
points in its vicinity upon the surface of the thorax, two chang
in the quality and pitch of the percussion note are observ
As the borders of the heart which are covered by the anteri
borders of the lungs are approached, the normal vesicular re
nance becomes impaired; and, finally, when the portion of t
right ventricle which lies in direct apposition with the chest w
is reached, the note becomes frankly flat. In this manner th
may be defined upon the anterior surface of the thorax two are
of cardiac dullness, the one within the other. The inner ar
representing the region in which the heart is uncovered by t
anterior borders of the lungs, is termed the *area of absolute c
diac dullness,* or the *area of cardiac flatness;* whereas, the peri
eral area, representing the portion of the heart which is overlapp
by the anterior pulmonary borders, is termed the *area of relat
cardiac dullness.*

The *area of absolute cardiac dullness,* representing the limit
portion of the right ventricle which is in direct apposition w
the anterior thoracic wall, corresponds to a roughly triangul
area, which is bounded upon the right by a vertical line dra
along the left border of the sternum from the level of the four

costal cartilage to the upper border of the sixth costal cartilage, and upon the left by a line drawn downward and outward from the junction of the fourth left costal cartilage with the sternum to the upper border of the sixth rib in the left parasternal line, corresponding to a point approximately one-half inch internal to the cardiac apex. Inferiorly the area of absolute cardiac dullness is continuous with the area of flatness of the liver.

The *area of relative cardiac dullness,* representing the portion of the heart which is overlapped by the anterior borders of the lungs, is bounded upon the right by a vertical line drawn upon the chest wall from the upper border of the third costal cartilage near its junction with the sternum along the right sternal border to the upper border of the sixth costal cartilage, and upon the left by a slightly curved line with its convexity directed toward the left and upward, drawn from the third left chondrosternal junction to the fifth intercostal space one-half inch internal to the midclavicular line. Inferiorly the area of relative cardiac dullness blends with the area of relative hepatic dullness.

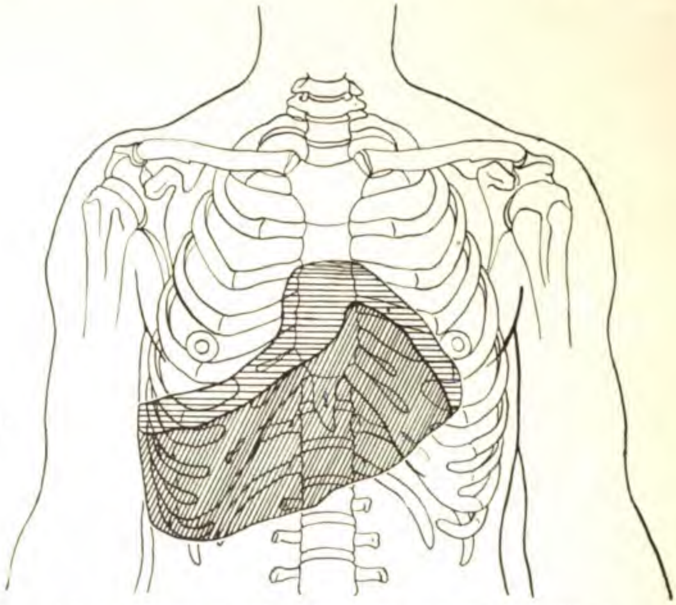


Fig. 135.—Areas of cardiac and hepatic dullness and flatness.

Thus, the areas of cardiac dullness roughly represent a triangle within a triangle, the area of absolute cardiac dullness lying within the confines of the area of relative dullness of the heart save inferiorly, where the boundaries are the same, and wher the flatness of the heart blends imperceptibly with that of th liver. Similarly, the right border of the area of relative cardia dullness joins the superior border of the area of hepatic dullnes at almost a right angle, the angle of pulmonary resonance in th fifth right interspace formed in this manner constituting *El stein's cardiohepatic angle.*

TECHNIC OF CARDIAC PERCUSSION

The areas of relative and absolute cardiac dullness, represent ing roughly the size, shape and position of the heart, may b outlined by ordinary mediate percussion, threshold percussio or auscultatory percussion.

In the practice of threshold percussion the pleximeter finge is bent at a right angle at the junction of the first and secon phalanges and the tip of the finger is closely applied to the inte costal space, avoiding contact with the ribs or costal cartilage The percussion blow is delivered lightly upon the first phalan thereby setting up vibrations in a very restricted area of th thorax.

Whatever method of percussion is employed, the examine should commence to percuss from three directions in order t fix the superior and lateral borders of the heart, employing a he proceeds both deep and superficial percussion.

In fixing the right and left borders, the examiner should pe cuss from the right and left axillary regions upon either side the thorax in the third, fourth and fifth interspaces toward th precordia, employing relatively deep percussion until impairme of resonance indicates that the lateral borders of the area relative cardiac dullness have been attained. Having marke these points upon the thoracic surface, the examiner should co tinue the percussion along the same lines, substituting superfici percussion for the deep strokes heretofore employed until th note changes to flatness, indicating that the lateral margins the area of absolute cardiac dullness have been reached, repr senting the portion of the right ventricle which is in direct a position with the anterior thoracic wall.

After having marked these several points upon the surface the thorax, the examiner, beginning in the left infraclavicul region, should percuss downward along the interval between th left sternal and parasternal lines, employing in the first instan deep percussion; and, when the superior limit of the area of relative dullness has been attained, he should continue with superficial percussion until the upper limit of the area of absolute cardiac dullness is reached, which is marked upon the surface of the thorax.

By connecting the points upon the chest wall at which the initial change in the percussion note was observed in each instance, the areas of relative and absolute cardiac dullness are graphically represented upon the surface of the thorax.

The delimitation of the dullness of the heart by percussion affords data sufficiently accurate for routine clinical work; but the cardiac outline thus obtained never represents the exact size and position of the heart. As a rule, the outline obtained errs one to two centimeters to the left of the right lateral border, while there is usually a like error toward the left in delimitation of the left lateral border of the heart. Over the superior sternal region it is not possible to distinguish between the areas of cardiac and vascular dullness by percussion.

In the practice of cardiac percussion, the examiner should utilize as far as possible the information furnished by the progressive increase of resistance which is encountered as the cardiac borders are approached. Piorry, Traube and Ebstein have each in turn emphasized the importance of this factor; and, indeed, Ebstein developed a special method of delimitation of the heart based upon the information thus obtained.

When for any reason the findings upon percussion of the precordia are obscure, resort should be had to the modern methods of fluoroscopy of the heart.

VARIATIONS IN THE AREAS OF CARDIAC DULLNESS

The areas of cardiac dullness present certain variations in position and outline depending upon the age of the subject, the posture assumed by the patient, the contour of the thorax, the position of the diaphragm, and the presence or absence of disease of the heart, pericardium or adjacent viscera.

During childhood the area of relative cardiac dullness is more extensive than at any later period of life. Owing to the greater flexibility of the thorax and the great elasticity of the lungs obtaining at this time of life, a relatively greater portion of the heart is exposed to the thoracic wall. Moreover, in the child the heart occupies a higher position in the thoracic cavity than is the case in the adult subject.

In children it is not uncommon to find the superior limit of the area of cardiac dullness occupying the second interspace in the left parasternal line, the left border extending to the midclavicular line in the third interspace, and surpassing this line by one-eighth to one-half inch in the fourth interspace, the point corresponding to the apex of the heart in this class of subject. Similarly, during childhood the area of absolute cardiac dullness is increased in a transverse direction, and is raised to the extent of one intercostal space.

In the aged subject, on the contrary, with the physiologic ptosis of the heart and abdominal viscera obtaining at this time of life, the areas of cardiac dullness occupy a lower level, the superior limit approximating the lower border of the fourth rib in the left parasternal line, while the left border approaches more nearly the left sternal border.

The dimensions of the areas of cardiac dullness vary with the posture assumed by the patient. In the recumbent posture the heart is in relation with a more extensive portion of the anterior thoracic wall, and the area of dullness as elicited upon percussion is extended to a corresponding degree. Upon assuming the right lateral decubitus, the area of dullness is displaced toward the right side of the thorax. Under these circumstances the area of cardiac flatness adjacent to the left sternal border is diminished or disappears entirely, while the area of dullness is distinctly extended to the right of the sternum. Upon assuming the left lateral decubitus, the area of cardiac flatness is increased transversely, and the area of cardiac dullness is carried approximately to the left midclavicular line.

In the robust subject with a deep thorax the areas of cardiac dullness are diminished in extent as outlined by percussion; whereas, in the case of the flat expiratory type of thorax in the normal subject, and in the presence of deficient inflation of the anterior pulmonary borders, the areas exhibit a corresponding increase in extent.

Depression of the diaphragm incident to advancing age or increased intrathoracic pressure is attended by a descent of the areas of cardiac dullness, while upward displacement of the diaphragm incident to increased subphrenic pressure is attended by elevation of the areas of dullness of the heart.

In the presence of disease of the heart, pericardium or other adhesions tends to draw the heart toward the side of the diseased pleura, with consequent shifting of the area of cardiac dullness. Subphrenic pressure in ascites, tympanites, hepatic enlargement or abscess, causes displacement of the area of cardiac dullness upward and toward the left side of the thorax. In all of these conditions the entire area of cardiac dullness is displaced; but, in the absence of coincident disease of the heart or pericardium, it is of normal dimensions.

Upward Increase.—Extension of the area of cardiac dullness in an upward direction accompanies pericarditis with effusion, and in the presence of aneurysm of the ascending portion or arch of the aorta a similar extension upward is observed. In pericarditis with effusion the area becomes irregularly triangular in outline, with the base directed downward as a result of the characteristic configuration of the pericardial sac.

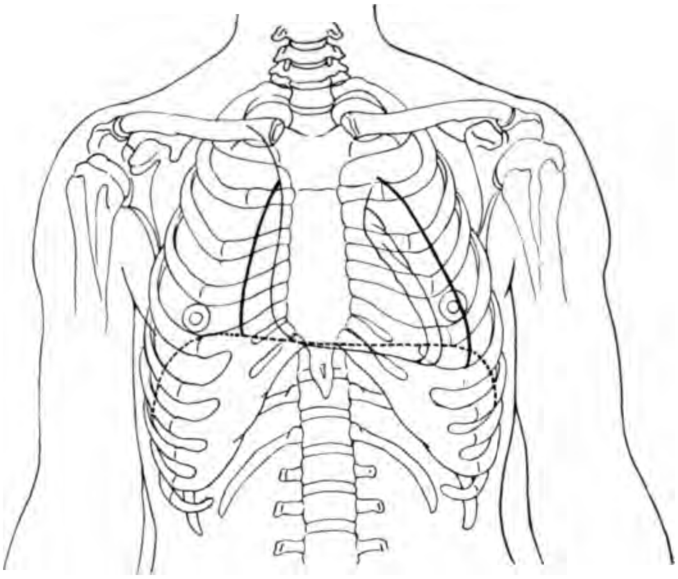


Fig. 137.—General extension of cardiac dullness in extensive pericardial effusion.

Increase to the Left.—An increase in the area of cardiac dullness toward the left occurs with hypertrophy and dilatation of the left ventricle, and in cardiac displacement by mediastinal pressure. In left ventricular hypertrophy the area is increased downward as well as toward the left, occasionally reaching the seventh interspace in the anterior axillary line.

Increase to the Right.—Extension of the area of cardiac du

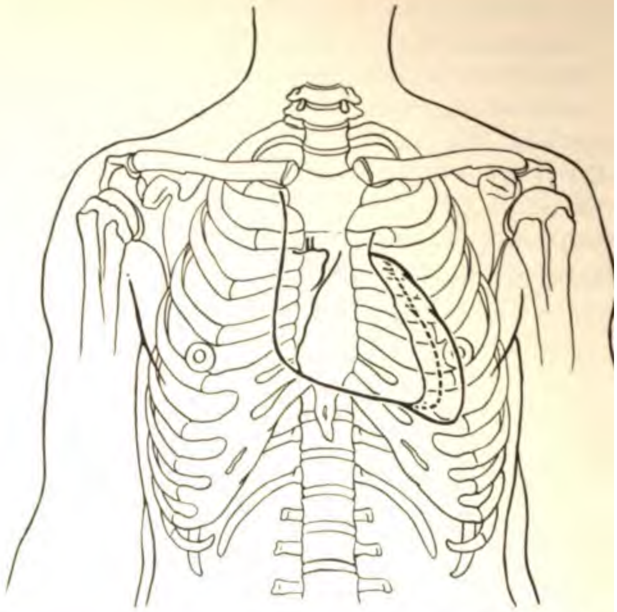


Fig. 138.—Extension of cardiac dullness toward the left and downward in lar hypertrophy. Dotted lines indicate normal cardiac outline.

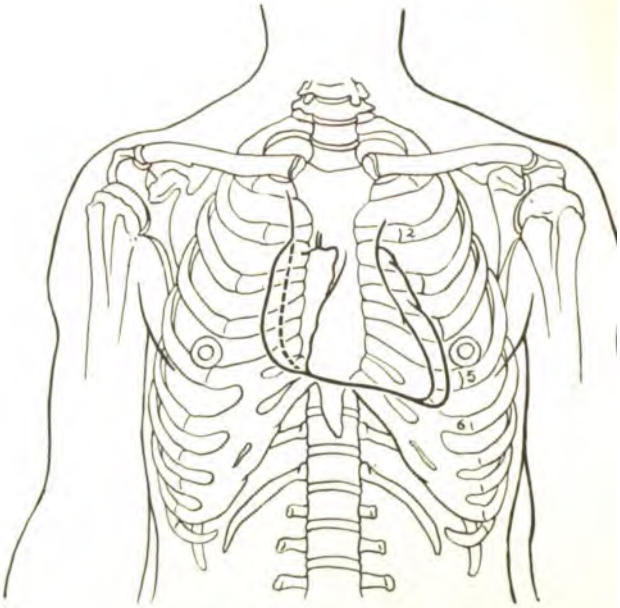


Fig. 139.—Extension of cardiac dullness toward the right in right vent trophy. Dotted lines indicate normal cardiac outlines.

ness toward the right, the dullness of the heart encroaching upon the normal vesicular resonance of Ebstein's cardiohepatic angle, occurs with right ventricular and auricular hypertrophy and dilatation, and in pericarditis with effusion, constituting in the latter disease *Rotch's sign.* A distended inferior vena cava may be responsible for a slight extension of the area of dullness to the right of the sternum. When the extension is the result of right ventricular hypertrophy there is frequently pulsation of the epigastrium, and not infrequently systolic pulsation of the jugular veins as a result of tricuspid regurgitation.

AREA OF VASCULAR DULLNESS

The area of vascular dullness, overlying the great vessels springing from the base of the heart, occupies the sternal region from the episternal notch to the level of the third costal cartilage. In the lower portion of this region the dullness of this area blends imperceptibly with the superior limit of the area of cardiac dullness.

An extension of this area of dullness toward the right in the first and second intercostal spaces occurs with aneurysm of the ascending aorta. Under these circumstances a visible or palpable pulsation of the subclavian arteries is frequently encountered in the supraclavicular regions, *Potain's sign.*

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An extension of this area of dullness toward the right in the first and second intercostal spaces occurs with aneurysm of the ascending aorta. Under these circumstances a visible or palpable pulsation of the subclavian arteries is frequently encountered in the supraclavicular regions, *Potain's sign.*

CHAPTER XV

AUSCULTATION

Object and Technic.—Auscultation is employed in the study of the circulatory organs to determine the intensity, quality and pitch of the sounds of the heart, their rhythm, and the presence or absence of certain adventitious sounds arising in the heart, pericardium and arteries.

During auscultation of the precordia mediate auscultation is to be employed in preference to immediate auscultation. During the examination no article of wearing apparel should intervene between the integument of the subject and the bell of the stethoscope. The attitude of the subject should be free from muscular tension; and, owing to the influence of the bodily attitude upon certain of the phenomena, the examination should be conducted with the subject in the erect and in the recumbent posture. In a limited number of cases, on account of the urgent dyspnea attending the underlying lesions, the examiner is forced to dispense with the examination in the recumbent posture.

In the examination of the sounds of the heart, auscultation is not practiced over the anatomic sites of the several valves, but in certain definite areas of the thorax in which the different heart sounds are audible with the maximum intensity and purity.

Auscultatory Valve Areas.—Each of the four valves of the heart has a corresponding area upon the thoracic surface at which the sound produced by closure of the valve in question is more distinctly audible than elsewhere. As stated, these areas do not correspond to the points upon the thoracic wall which are nearest to the anatomic sites of the valves.

The sound produced by closure of the mitral valve is most clearly audible in the *mitral area*, which is situated in the fifth left intercostal space over the apex of the heart, although the anatomic site of this valve is behind the left half of the sternum at the level of the fourth costal cartilage.

The sound produced by closure of the aortic valve is most clearly audible at the *aortic area*, which is situated in the second intercostal space immediately adjacent to the right sternal border, although the anatomic site of this valve is posterior to the left half of the sternum at the level of the third interspace.

The *tricuspid area,* at which sounds arising from the tricu valve are most distinctly audible, is situated over the lower tion of the sternum, the anatomic site of this valve being situ behind the right portion of the sternum between the level of fourth and sixth costal cartilages.

The *pulmonary area,* at which sounds generated by the actio the pulmonary valve are most distinctly audible, occupies a p immediately to the left of the sternum in the second interc space, the anatomic site of this valve being posterior to junction of the third left costal cartilage with the sternum.

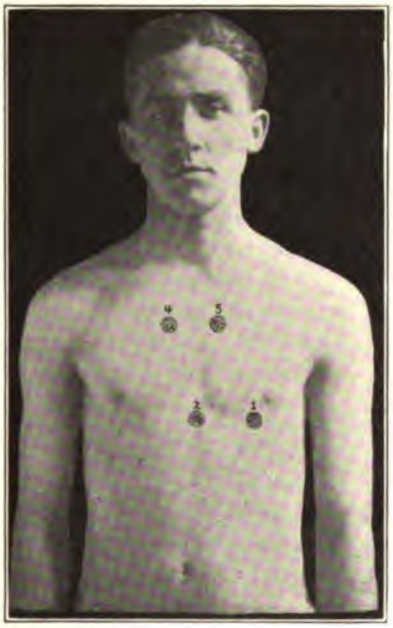


Fig. 140-*A*.—Auscultatory valve areas of the heart. *1*, Mitral area; *2*, tricuspid area; *3*, pulmonary area; *4*, aortic area.

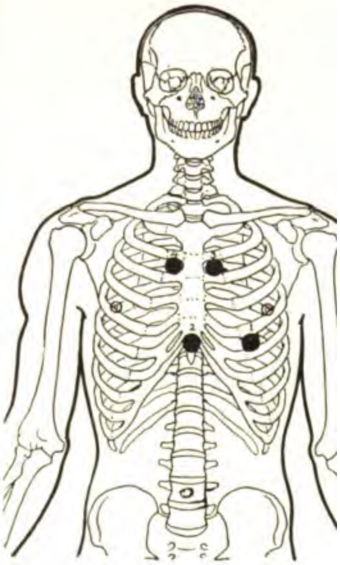


Fig. 140- *B*.—Auscultatory valve ar the heart. *1*, Mitral area; *2*, tricuspid *3*, pulmonary area; *4*, aortic area.

The Normal Heart Sounds.—The heart in its rhythmic ac produces two sounds, which are termed respectively the *first* the *second* sound of the heart. The first sound is audible clearly in the region of the cardiac apex in the normal sub while the normal second sound is most clearly audible at the of the heart.

The *first sound* of the heart is audible during ventricular tole; it is lower in pitch and of longer duration than is the se sound; and it has been compared acoustically to the soun the word "lubb." The first sound is produced by the systolic contraction of the ventricles, the vibration of the auriculoventricular valves upon closure, and the sudden distention of the proximal portions of the aorta and pulmonary artery. It is a combined muscular and valvular sound, and a careful study of the two essential elements of the sound is of service in drawing conclusions as to the integrity of the myocardium.

The *second sound* of the heart is audible during the early portion of ventricular diastole. The sound marks the beginning of diastole; but it consumes only the early portion of diastole, hence is really protodiastolic in time. The second sound is of higher pitch and distinctly shorter duration than is the systolic sound; and it has been likened acoustically to the sound of the word "dup." The second sound of the heart is produced by the sudden coaptation of the segments of the semilunar valves, which guard the orifices of the aorta and the pulmonary artery.

Occasionally there is to be detected a *third sound* of the heart, occurring approximately 0.1 second after the diastolic sound. Thayer attributes the third sound to sudden tension of the mitral valve, occurring with the entrance of the blood at the commencement of diastole. The sound is most frequently to be detected in children during periods of bradycardia with the subject in the left lateral decubitus.

Intensity of the Heart Sounds.—Upon auscultation of the precordia of the normal subject, it is noted that the individual sounds arising at the different valve areas of the heart are not of uniform intensity. Thus, although the first sound of the heart is produced by the combined action of the ventricles and the auriculoventricular valves, it is observed that the first sound at the mitral area is lower in pitch and of somewhat greater duration than is the tricuspid first sound. Similarly, in the examination of the component valves concerned in the production of the second sound, it is observed that in the adult subject the second sound at the aortic area is of greater intensity than is the second sound generated by closure of the pulmonary valve; whereas, in the child the condition is reversed, the pulmonary sound exceeding in intensity the aortic second sound.

VARIATIONS OF INTENSITY

The intensity of the cardiac sounds, as elicited upon auscultation of the several valve areas, varies in normal subjects of different types, with variations in the thickness of the thoracic wa with the state of the subjacent pulmonary tissues, and with enc cardial and exocardial disease. A correct evaluation of the i tensity of the heart sounds can only be made after extensi clinical experience. Variations in intensity may involve one both sounds of the heart, and the change in intensity frequent is associated with a definite change in the quality and pitch the sound.

Accentuation of Both Sounds.—Both first and second sounds the heart exhibit accentuation following the ingestion of stim lants, and during physical effort or emotional excitement. T accentuation in this instance is transient, and is devoid of pat ologic significance. A more permanent accentuation occurs wi cardiac hypertrophy, during exophthalmic goiter, and in the pr ence of acute febrile diseases.

An apparent accentuation of both cardiac sounds is occasional encountered in subjects with very thin chest walls, and in patier in whom fibroid retraction of the anterior border of the left lu exposes the heart freely to the anterior thoracic wall. Similar consolidation or carcinomatous infiltration of the lappet of lu overlying the heart transmits the normal cardiac sounds to t surface of the thorax with undue intensity, simulating a tr accentuation of the tones.

Diminished Intensity of Both Sounds.—In the robust su ject with a deep thorax, and in the obese subject with thi subcutaneous tissues, there is a pseudo-diminution of t intensity of the heart sounds upon auscultation of the p cordia. Similarly, in hypertrophic emphysema, when the d tended anterior borders of the lungs intervene between the hea and thoracic wall, the cardiac sounds are enfeebled without p sessing any reference to the state of the myocardium. In t presence of moderate pericardial effusion, when a limited amou of fluid intervenes between the heart and the chest wall, there noted a diminution in the intensity of both sounds.

In the presence of myocardial degeneration and cardiac dila tion, however, the cardiac sounds are enfeebled as a result impairment of the integrity of the myocardium. General thenia from chronic wasting disease is likewise attended diminution of the intensity of the heart sounds.

Accentuation of the First Sound.—An increase in the intens of the first sound of the heart at the mitral area, of moderat increased duration, followed by an accentuated aortic sound, indicative of left ventricular hypertrophy. When cardiac dilatation is imminent, the first sound as elicited at the apex is loud, but is of brief duration and has engrafted upon it the valvular quality of the normal second sound of the heart.

During the course of acute sthenic fevers, when the ventricle is contracting powerfully in the presence of low arterial tension, the first sound of the heart at the apex is accentuated. In exophthalmic goiter the first sound is likewise increased in intensity, but with a diminution in the muscular element and an increase in the valvular quality of the sound.

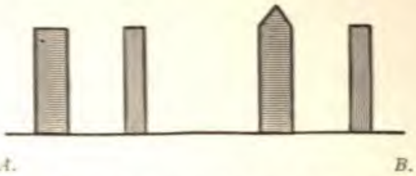


Fig. 141.—*A*, Normal first and second sounds. *B*, Accentuated first sound.

Diminution of the First Sound.—Myocardial degeneration and cardiac dilatation are attended by diminution in the intensity of the first sound at the apex. Similarly, the asthenia of chronic wasting disease, anemia, and prolonged fever results in diminution in the first sound, the tone acquiring a valvular quality analogous to that of the normal second sound of the heart.

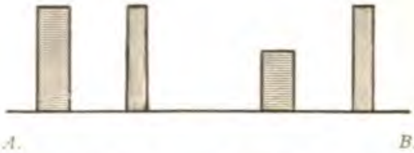


Fig. 142.—*A*, Normal first and second sounds. *B*, Diminished first sound.

Accentuation of the Aortic Sound.—The aortic sound is accentuated in all physical states which are attended by increased tension in the greater circulation. Hence, it is encountered in cases of angiospasm due to transient vasoconstriction, in arteriosclerosis, chronic nephritis, uremia, and in apoplexy. When the proximal portion of the aorta and the semilunar valves participate in general arteriosclerosis, the aortic sound is accentuated, metallic and ringing. Pregnancy is attended by moderate accentuation of the aortic sound of the heart.

In left ventricular hypertrophy arising as a sequence of arteriosclerosis or chronic hypertension in the greater circulation, there is coincident accentuation of the aortic sound and of the first sound at the apex. With the inception of cardiac dilatation under these circumstances the first sound at the apex becomes progressively enfeebled, with maintenance of the accentuation of the aortic sound. Such a change in the relative intensity of the two sounds is always of grave prognostic import.

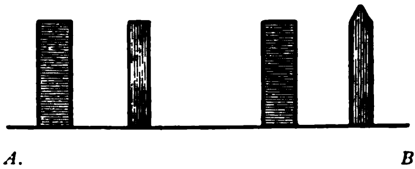


Fig. 143.—*A*. Normal first and second sounds. *B*. Accentuated second sound.

Diminution of the Aortic Sound.—Enfeeblement of the aortic sound accompanies lowering of the blood pressure in the greater circulation incident to profuse hemorrhage, anemia, relaxation of the peripheral arterioles, and when, as a result of mitral or aortic stenosis or insufficiency, a diminished quantity of blood is ejected into the aorta during ventricular systole. The aortic sound is likewise diminished in intensity in the presence of myocardial degeneration and dilatation of the left ventricle, unattended by hypertension in the greater circulation.

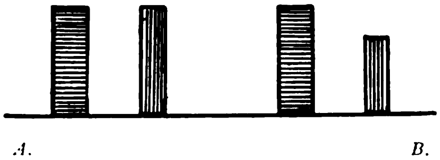


Fig. 144.—*A*. Normal first and second sounds. *B*. Diminished second sound.

Accentuation of the Pulmonary Sound.—Hypertension in the pulmonary circulation is attended by accentuation of the pulmonary sound of the heart. Such hypertension may be of endocardial or of exocardial origin. Regurgitant and stenotic lesions at the mitral or aortic valve, by causing the blood to accumulate in the pulmonary circuit, result in accentuation of the pulmonary sound.

Pulmonary hypertension of exocardial origin occurs in the presence of obstructive pulmonary disease, and in mechanical congestion of the lungs induced by the pressure of tumors or enlarged glands upon the great veins returning blood from the lungs to the left auricle. Accentuation of the sound due to pulmonary disease is encountered during the course of phthisis, pneumonia, hypertrophic emphysema and cirrhosis of the lung.

In the presence of right ventricular hypertrophy arising from any cause, associated with integrity of the pulmonary valve, the second sound is accentuated at the pulmonary area.

Diminution of the Pulmonary Sound.—Diminution in the intensity of the pulmonary sound is indicative of imminent failure of the right ventricle. When, during the course of left-sided valvular disease or obstructive disease of the lungs, a pulmonary sound which has shown accentuation becomes enfeebled, it indicates right ventricular dilatation or the incidence of tricuspid regurgitation.

Quality of the Heart Sounds.—The low-pitched, muscular booming quality essentially belongs to the first sound of the heart, whereas the high-pitched, valvular quality is distinctive of the second sound. In the presence of myocardial degeneration, when the ventricular contractions are increased in frequency, there is a diminution in the muscular element of the first sound at the apex, the sound acquiring a valvular quality analogous to that of the normal second sound. Similarly, in exophthalmic goiter there is noted a diminution in the muscular element and an assumption of the valvular quality by the first sound at the apex.

When arterial sclerosis involves the proximal portion of the aorta and the semilunar valves, the second sound of the heart at the aortic area has imparted to it a loud, metallic and ringing quality, quite dissimilar to the normal second sound. Similarly, in the presence of extensive pneumothorax or gastric distention, and when an extensive pulmonary excavation adjacent to the heart contains air, the cardiac sounds have a metallic quality of exocardial origin imparted to them.

Pitch of the Heart Sounds.—During the course of acute febrile diseases, and in the presence of acute myocarditis, the first sound of the heart as appreciated at the apex is increased in pitch, the change in pitch being associated with a diminution in the duration of the sound. Increased pitch of the second sound is encountered at the aortic or pulmonary areas when the pressure is raised in the general or pulmonary circulations. Elevation of the pitch of the sounds also attends the alteration in quality which is noted when large air-containing cavities occupy regions of the thorax or abdomen adjacent to the heart.

Duration of the Heart Sounds.—In the presence of hypertensi affecting the greater circulation, attended by adequate comp sation by the left ventricle, the first sound of the heart at t apex is moderately prolonged, the muscular element of the sou to a great degree obscuring the valvular element. Palpation the mitral area under these circumstances reveals the presence a strong, sustained and heaving cardiac impulse of considera lifting power.

In the presence of myocardial degeneration, on the contra there is noted a diminution in the duration of the first sound the heart. The first sound under these circumstances is likew diminished in intensity, is raised in pitch, and has engraft upon it the valvular quality of the normal second sound of t heart.

REDUPLICATION OF THE HEART SOUNDS

Either the first or the second sound of the heart may un appropriate conditions become doubled or reduplicated. Usua the examiner encounters a reduplication of the first or seco sound alone, but occasionally there is to be detected a redupli tion of both sounds of the heart.

Reduplication of the first sound of the heart is manifest acoustically by a sound resembling the words "lur-rup-dup." T adequate solution of the mode of production of the sound is be with difficulties. It has been attributed to asynchronous clos of the mitral and tricuspid valves, arising as a result of as chronous systole of the right and left ventricles. The p nomenon has also been ascribed to unequal tension of the leafl of the auriculoventricular valves. It has likewise been suggest that the phenomenon is caused by tardy contraction of the pa lary muscles, as these muscles are supplied by terminal arter and are readily subject to fatigue.

The sound produced by reduplication of the first sound of t heart is readily confused with a presystolic mitral murmur wh this is followed by a normal first sound of the heart. The sou which is commonly limited to the apical area, has been noted mitral stenosis, myocardial degeneration, arteriosclerosis, a chronic adhesive pericarditis.

Reduplication of the second sound has been compared acou cally to the sound of the spoken words "lub-durrup." The p nomenon is attributed to asynchronous closure of the aortic a pulmonary valves, occurring as a result of unequal tension in the greater and lesser circulations. Under these circumstances the ventricle which has to overcome the heightened pressure contracts more slowly than its fellow and the semilunar valves close at an appreciably later period than do those of the corresponding valve of the opposite side.

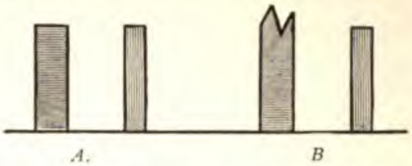


Fig. 145.—*A*. Normal first and second sounds. *B*. Reduplicated first sound.

This circulatory imbalance occurs in all states which raise the pulmonary blood pressure, such as hypertrophic emphysema, cirrhosis of the lung, the consolidations of pneumonia and phthisis, and left-sided valvular lesions of the heart. Similarly, the pressure is raised in the general circulation in the presence of arteriosclerosis, temporary angiospasm, and chronic nephritis.

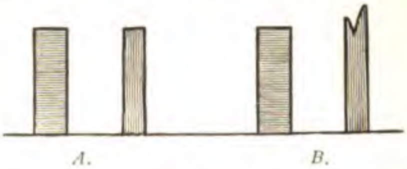


Fig. 146.—*A*. Normal first and second sounds. *B*. Reduplicated and accentuated second sound.

Reduplication of the second sound has occasionally been noted in the normal subject during forced inspiration.

GALLOP RHYTHM

The introduction of a third sound into the cardiac cycle in certain cases of cardiac asthenia and cardiovascular disease constitutes the *gallop rhythm* of Potain. The superadded sound may occur just prior to ventricular systole, or it may be audible during the early portion of ventricular diastole.

Presystolic gallop rhythm is characterized by the occurrence of a third sound which is audible just prior to the first sound of the heart, simulating acoustically the presystolic murmur. It has been noted especially in association with mitral stenosis, and has been ascribed to undue force of the auricular systole. The sound has also been attributed to asynchronous contraction of the rig and left ventricles. Aside from mitral stenosis, presystolic gall rhythm has been noted in association with hypertension in t greater circulation with imminent failure of the left ventric and during the asthenia of prolonged infections.

Diastolic gallop rhythm is to be considered merely an exa geration of the third sound of the heart, and is to be attribut to the sudden tension of the mitral cusps upon the inrush of t auricular blood column during early diastole. Occurring in co ditions associated with cardiac asthenia, the later during di tole the sound has its inception, the more grave is the progno

ADVENTITIOUS SOUNDS

In the presence of disease of the circulatory organs various a ventitious or new sounds are generated within the chambers the heart (endocardial), or in the pericardium, lungs, pleura vessels (exocardial).

Adventitious sounds comprise endocardial murmurs, the c diorespiratory murmur, pericardial friction, the pericardial s cussion sound, and murmurs arising in the arteries and veins

ENDOCARDIAL MURMURS

Endocardial murmurs are adventitious sounds arising witl the chambers of the heart or in the proximal portions of the gr arteries springing from the ventricles. They may be superadd to the cardiac sounds, or they may entirely replace the soun of the heart.

The manner in which endocardial murmurs are generated m be explained upon certain definite physical principles. Wher circulating fluid medium passes at a uniform velocity througl tube of uniform calibre no sound is produced. But when t tube is constricted at one point in such manner that the circul ing fluid passes through a constricted orifice into a wider port of the tube, vibrations or eddies are engendered in the flu which are attended by audible sound.

Similarly, endocardial murmurs are produced by irregularit in the movement of the blood through the chambers and orifi of the heart, by virtue of which the volume of circulating bl is thrown into vibration with the production of eddies or "fl veins," which become audible as murmurs and palpable as thri

As long as the normal quota of blood, of normal density, passes through a normal heart with intact endocardium and valves, no sound is generated save the normal heart sounds. But when the blood is forced through a narrowed or stenotic orifice into a wider chamber beyond, or when the blood is permitted by an incompetent valve to regurgitate into a chamber of the heart, an endocardial murmur becomes audible upon auscultation.

In the presence of dilatation of the ventricle the muscular ring of the auriculoventricular valve is prone to yield to such a degree that the normal valve cusps are incapable of occluding the abnormally large orifice, resulting in a condition of relative insufficiency of the valve. Similarly, in the case of the aortic and pulmonary valves, dilatation of the proximal portion of the aorta or of the pulmonary artery may so alter the relation of the size of the normal orifice to the arterial caliber as to produce fluid veins in the artery with the production of a murmur in the absence of stenosis of the orifice.

The density of the blood also influences the generation of endocardial murmurs. In the presence of grave anemia the density of the blood is frequently reduced to such a degree that the blood column is whipped into eddies while passing through normal orifices into chambers of normal caliber, producing *hemic* or functional *murmurs* in the absence of disease of the heart.

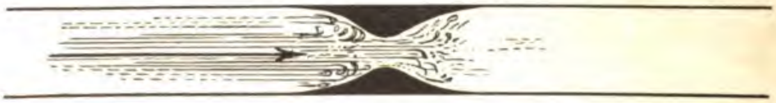


Fig. 147.—Illustrating the physical basis of murmurs generated by diminution of lumen.

A certain degree of endocardial pressure is essential to the generation of endocardial murmurs. This is evinced by the fact that endocardial murmurs remain distinctly audible as long as cardiac compensation is maintained, and become indistinct or lost with the supervention of cardiac dilatation.

CHARACTERISTICS OF ENDOCARDIAL MURMURS

Skoda first called attention to the diagnostic importance of the characteristics or properties of endocardial murmurs, and showed that by the detailed study of these several characteristics murmurs arising at the several orifices of the heart may be isolated, and their site of production may be determined.

Intensity.—The intensity of a murmur is dependent upon th state of the myocardium, the velocity of the circulating bloo stream, the size of the orifice and the dimensions of the cavit of the heart in question, and the density of the blood.

Just as a certain degree of endocardial pressure is essenti to the generation of a murmur, so also is the intensity of th murmur a good index to the state of the myocardium. Thus, loud murmur suggests the presence of cardiac hypertrophy, whil a faintly audible murmur is very suggestive of inadequate con pensation. Moreover, a change in the intensity of a murmu during daily examinations affords an index to the reserve powe of the heart, a change from a loud to a soft, faintly audible mu mur suggesting a failing heart, whereas a progressive increas in the intensity of a murmur from day to day is suggestive c cardiac improvement.

In the case of systolic murmurs, in the generation of which th blood stream is propelled with considerable force by the ve tricular contractions, the intensity is usually greater than tha of diastolic murmurs, in the generation of which the blood strea is propelled by the elastic recoil of the aorta or pulmonary arter Similarly, in the case of presystolic murmurs the blood strea is not propelled by auricular systole with the same degree c force as in the case of systolic murmurs engendered during ve tricular systole.

In general, the intensity of a murmur is in direct proportion t the size of the orifice at which it is produced and the dimensior of the cavity into which the blood column is propelled; namel the greater the degree of stenosis, the greater the intensity of th murmur which is generated at the orifice in question. Howeve in drawing conclusions as to the degree of stenosis from th intensity of the murmur, the examiner should consider its dur tion and quality, the state of the myocardium, and the extraneou factors which may influence the intensity of endocardial mu murs.

The posture of the patient exerts a striking influence upon th intensity of endocardial murmurs. In general, regurgitant mu murs are most intense when the subject assumes the dors decubitus, while stenotic murmurs exhibit their maximum inte sity with the patient in the erect posture. The influence o posture upon the intensity of murmurs is exhibited chiefly i the case of murmurs arising at the mitral and tricuspid valve however, as murmurs generated at the aortic and pulmonary valves are only slightly influenced by these changes.

In the presence of grave anemia an endocardial murmur is apt to exhibit an increase in intensity, and under these circumstances the quality of a murmur is frequently altered.

The intensity of endocardial murmurs is influenced by the depth of inspiration, as well as by the state of the superimposed pulmonary tissues and the chest wall. Upon forced inspiration the anterior borders of the lungs cover an extensive portion of the heart and diminish the intensity of endocardial murmurs, as appreciated upon auscultation of the precordia. Similarly, the voluminous anterior pulmonary borders in hypertrophic emphysema, by intervening between the heart and chest wall, diminish the intensity of sounds emanating from the chambers of the heart. In the robust subject with deep thorax, in the obese subject with thick chest wall, and in the thorax which is the seat of osseous deformity the intensity of endocardial murmurs is masked without reference to the state of the myocardium. The presence of large air-containing cavities adjacent to the heart, on the contrary, and the presence of extreme tympanites are capable of producing a false augmentation in the intensity of endocardial murmurs.

Physical exertion increases the intensity of endocardial murmurs, and judicious exercise frequently reveals the presence of an obscure murmur, which is not detected during examination in repose.

Point of Maximum Intensity.—Every endocardial murmur has a point of maximum intensity, the point at which it is most distinctly audible upon auscultation of the thorax. These points correspond very accurately, on the whole, with the points at which the closure of the normal valves are most clearly audible; namely, in the four acoustic valve areas. Thus, a murmur which is generated at the mitral valve is usually heard with the greatest intensity at the mitral area, over the apex of the heart in the fifth left interspace; whereas, a murmur which is generated at the pulmonary valve is most clearly audible at the pulmonary area in the second interspace adjacent to the left sternal border.

This selective transmission of the sound in its maximum intensity to the surface of the thorax in the case of the different murmurs is accounted for by the fact that the sound is conducted most readily in the direction of the blood current which called

it into being, and by differences in the conductivity of the co
ponent portions of the heart and of the thorax.

Line of Transmission.—The majority of endocardial murmu
are audible not only at their points of maximum intensity, b
are transmitted thence in directions which vary in the individu
murmurs, constituting the line of transmission or area of prop
gation of the murmur. The direction in which a given murm
is to be transmitted is determined in certain instances by t
direction of the blood stream, in other cases by the arrangeme
of the papillary muscles and ventricular wall, and again by t
relations of the several chambers of the heart to the lun
mediastinal structures and the chest wall.

The distance to which a murmur is transmitted is depende
upon the state of the myocardium, whether hypertrophied
the seat of retrogressive changes, the initial intensity of t
murmur, the size of the heart, and the state of the superimpos
pulmonary tissues and chest wall.

Thus, with augmentation of the propulsive force of the l
ventricle in hypertrophy of this chamber, and the increase
the size of the heart in massive hypertrophy, the systolic murm
of mitral regurgitation is transmitted well into the left axilla
region, and occasionally as far as the angle of the scapula.
compensation fails, the area of propagation of the murmur cov
an ever-diminishing area of the chest wall. Moreover, in t
case of the mitral systolic murmur the line of transmission does
follow the direction of the blood stream which calls the murm
into being, but is propagated toward the cardiac apex by t
anterior papillary muscles and the wall of the left ventricle.

Time of Murmurs.—Every endocardial murmur bears a defin
temporal relation to the events of the cardiac cycle, as they
generated during auricular systole, ventricular systole, or v
tricular diastole. A murmur which is generated by ventricu
systole is audible during this phase of the cardiac cycle and
termed a *systolic murmur;* while a murmur developing and audi
during ventricular diastole is designated a *diastolic murmur.* Si
ilarly, a murmur which is audible just prior to ventricular syst
or, in other words, during auricular systole, is termed a *presyst
murmur.*

Murmurs are further defined according to the portion of
individual phase of the cardiac cycle in which they are audi
Thus a *protosystolic* murmur is audible during the commencem

of systole, a *meso-* or *midsystolic* murmur is audible during the middle of systole, while a *telesystolic* murmur is audible toward the end of the systole.

At the bedside, the time of endocardial murmurs is confirmed by palpation of the cardiac impulse or of the carotid artery during auscultation of the precordia, or by noting the temporal relation of the murmur to the first sound or to the long pause of the heart.

Quality of Murmurs.—Endocardial murmurs have been variously described as harsh, coarse and rasping, or as soft, blowing and musical. The diagnostic value of the quality of murmurs has been the subject of warm discussion for many years. In general, harsh and unmusical murmurs accompany stenotic lesions, while soft, musical or blowing murmurs characterize regurgitant lesions of the cardiac valves. But in the evaluation of the quality of individual murmurs it is to be borne in mind that the quality of a murmur is subject to change from time to time with changes in the size and structure of the orifice and valve segments, the caliber of the chamber of the heart into which the blood column is propelled, the degree of hypertrophy which is present, and the presence or absence of anemia. Moreover, when a murmur is generated in the heart which is adjacent to extensive pulmonary excavations containing air or to pneumothorax, or even in the presence of extreme tympanites or gastric distention, the murmur is apt to assume an altered quality of exocardial origin.

While studying the quality of the murmur, the examiner should also endeavor to determine whether the murmur is followed by the normal cardiac sound or whether it replaces this sound, as a murmur which merely accompanies or is superadded to the normal cardiac sound is not ordinarily of as grave prognostic significance as is a murmur which entirely replaces the sound of the heart.

Duration of Murmurs.—Murmurs may occupy any portion of the phase of the cardiac cycle in which they occur; they may be followed by a normal cardiac sound or may replace this sound; and their duration is measured by their relation to the duration of the several phases of the cardiac cycle.

The duration of a murmur frequently gives a clue to the degree of stenosis or regurgitation which is present. In the presence of adequate compensation, a brief "whiff" at a valve area, the murmur occupying only the earlier portion of the phase of the cardiac cycle in which it is audible, points to rapid expulsion through a relatively wide orifice; whereas, a prolongation of the murmur, usually with maintenance of the intensity or a crescendo effect, points to slow expulsion of blood through a narrow or stenotic orifice.

MITRAL MURMURS

Organic murmurs are generated at the mitral valve in the presence of stenosis and incompetence of the valve; and they are presystolic and systolic respectively, as they are audible just prior to or during ventricular systole.

Mitral Presystolic Murmur.—A presystolic murmur at the mitral area is indicative of mitral stenosis, the narrowing of the orifice whipping the blood stream into eddies which produce a murmur, which is audible just prior to the first sound of the heart. The murmur is commonly followed by a sharp, snapping first sound; but as mitral stenosis and regurgitation frequently coexist, the regurgitant murmur often masks or replaces the first sound of the heart at the apex.

The point of maximum intensity of the murmur is localized in the mitral area in the fifth left interspace over the cardiac apex, whence the murmur is not transmitted. The mitral presystolic murmur is loud, harsh and crescendo in quality, increasing in intensity to its abrupt termination, usually in a sharp first sound of the heart.

The precise time of appearance of the murmur of mitral stenosis varies with the period of the disease. During the first stage of the disease, during the maintenance of left auricular hypertrophy, the murmur is generated immediately prior to ventricular systole; and the first sound of the heart at the apex, in the absence of coincident mitral regurgitation, remains clearly perceptible. During the second stage of the disease, with the establishment of right ventricular hypertrophy, the mitral murmur develops slightly earlier in diastole and is well sustained throughout auricular systole, with an intensity which equals or exceeds that of the murmur during the first period of the disease. With failure of the right ventricle the mitral murmur becomes progressively more feeble, to finally become inaudible or to be obscured by a systolic murmur at the tricuspid area, arising as a result of tricuspid regurgitation.

The murmur of mitral stenosis is quite constantly accompanied

by a palpable thrill over the cardiac apex; and the pulm
second sound is commonly accentuated as a result of incr
tension in the pulmonary circulation.

The murmur of mitral stenosis must be differentiated fro
Flint murmur, which is also audible at the mitral area just
to ventricular systole in cases of aortic regurgitation. The
ner of generation of this murmur has not been adequatel
plained; but the accepted explanation is that in this diseas
aortic cusp of the mitral valve becomes the target for two str

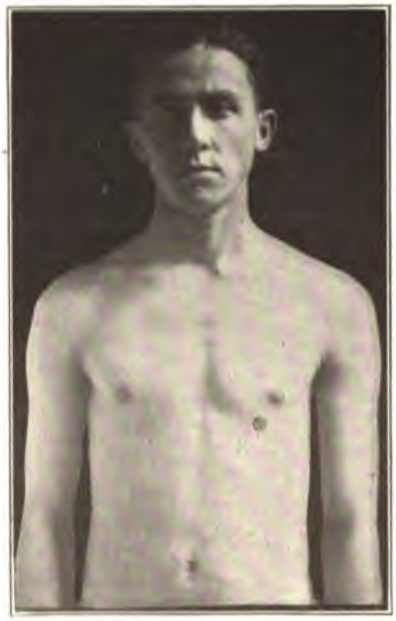


Fig. 148-*A*.—Point of maximum intensity of mitral presystolic murmur.

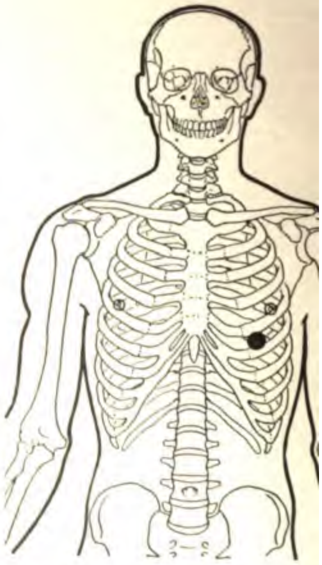


Fig 148-*B*.—Point of maximum in
of mitral presystolic murmur

of blood entering the left ventricle from opposite directions
one entering from the left auricle and the other regurgit
from the aorta, and is thereby thrown into vibrations, whicl
audible as a late diastolic or presystolic murmur.

The Flint murmur has its point of maximum intensity a
mitral area; it is audible during late diastole or just pri
systole; and it is not transmitted from the apical area. B
has not the ingravascent or crescendo quality of the mitral
otic murmur; it is not followed by a snapping first sound o
heart; it is not accompanied by a palpable thrill; and it has

ciated with it other signs of aortic regurgitation, as pulsation in the arteries of the neck, the water-hammer pulse, and occasionally the capillary pulse of Quincke.

In acute fibrinous pericarditis there is occasionally an audible presystolic friction sound which may be mistaken for the presystolic mitral murmur. Careful auscultation of the precordia will usually demonstrate that the point of maximum intensity of this sound is situated above the level of the cardiac apex.

The adventitious sound introduced into the cardiac cycle in presystolic gallop rhythm may likewise be confused with the presystolic mitral murmur.

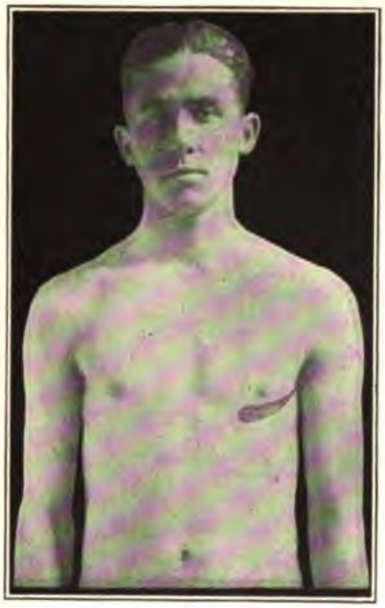


Fig. 149-*A*.—Point of maximum intensity and line of transmission of mitral systolic murmur.

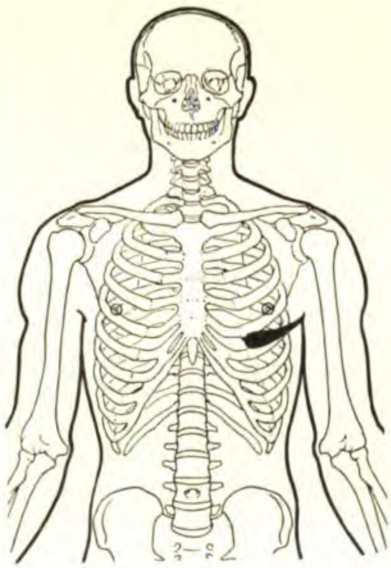


Fig. 149-*B*.—Point of maximum intensity and line of transmission of mitral systolic murmur.

Mitral Systolic Murmur.—Systolic murmurs audible at the mitral area fall within two classifications: namely, *organic* murmurs due to insufficiency of the mitral valve occurring as a result of organic deformity of the valve segments, or due to stretching of the mitral ring, whereby the borders of the cusps cannot be brought into close coaptation during ventricular systole; and *functional* murmurs, which have no reference to the state of the endocardium or valves of the heart. The two types of murmur present inherent characteristics which serve to differentiate the one from the other.

The point of maximum intensity of the organic mitral systolic murmur is localized in the mitral area over the apex of the heart, whence the murmur is transmitted toward the left axillary region, not infrequently as far as the angle of the scapula. In the case of this murmur the direction of transmission is not determined by the direction of the blood stream, which is regurgitating through the incompetent mitral orifice, but by the anterior papillary muscles and the wall of the left ventricle, which conduct the sound toward the apex of the heart.

The distance to which the murmur is transmitted is influenced by the size of the left ventricle and the transverse diameter of the thorax. Thus, the greater the hypertrophy of the left ventricle, and the more limited the transverse diameter of the thorax, the more nearly does the cardiac apex approach the lateral thoracic wall, and the farther toward the scapular angle is the murmur conducted.

The murmur occurs during ventricular systole. It may consume only the early or the latter portion of this phase of the cardiac cycle, or it may replace the first sound of the heart at the apex. In quality the murmur is commonly soft and blowing or musical and of low pitch. The intensity of the murmur varies with the state of the myocardium, remaining intense as long as compensation is maintained, and becoming less clearly audible or disappearing when dilatation is imminent or has occurred. The murmur is usually most clearly audible with the patient in the recumbent posture.

The murmur of mitral regurgitation is less constantly accompanied by a palpable thrill than is the murmur of stenosis of this valve.

The mitral systolic murmur of *relative mitral regurgitation*, occurring during the course of acute febrile diseases, anemic states, acute parenchymatous myocarditis, and after excessive physical effort, in which there is no structural deformity of the valve, but merely a stretching of the muscular ring at the base of the valve, is frequently evanescent in its manifestations, lacks the initial intensity of the organic mitral systolic murmur, is not attended by accentuation of the pulmonary second sound, and is not transmitted to any considerable distance from its point of maximum intensity.

The pulmonary second sound is accentuated in the majority of cases of organic mitral regurgitation, owing to right ventricular hypertrophy or occurring as a result of the heightened pressure in the pulmonary circulation; and, in long standing cases with marked changes in the mitral valve, a "safety-valve leak" frequently develops at the tricuspid valve.

AORTIC MURMURS

Murmurs generated at the aortic orifice are systolic and diastolic respectively, as they are occasioned by an obstruction to the free flow of blood from the ventricle into the proximal portion of the aorta during ventricular systole, or by lesions of the valve which by impairing its integrity permit a portion of the blood ejected during systole to regurgitate into the ventricle during diastole. Systolic murmurs occurring in the aortic area are likewise to be attributed in certain instances to aortic roughening, and to dilatation of the proximal portion of the aorta, in the absence of stenosis of the aortic valve.

Aortic Systolic Murmur.—Stenosis of the aortic orifice is attended by a systolic murmur of striking intensity and harsh, unmusical quality, with its point of maximum intensity in the aortic area, in the second intercostal space adjacent to the right sternal border. The murmur is conducted from its site of production to its point of maximum intensity by the blood stream in the ascending aorta. In the presence of extreme grades of stenosis of the aortic orifice, associated with excessive left ventricular hypertrophy, the intensity of the murmur is such that it is audible over the entire precordia, and may readily be mistaken for a systolic murmur arising from the mitral valve. Careful auscultation of the precordia, however, will reveal the point of maximum intensity of the murmur in the aortic area.

From its point of maximum intensity the aortic systolic murmur is transmitted by the arterial stream into the carotid and subclavian arteries. In the absence of coincident incompetence of the aortic valve the stenotic murmur is followed by a slightly muffled second sound at the aortic area, the muffling of the sound in this instance contrasting in a striking manner with the purity of the second sound in the presence of a systolic murmur generated by changes in the proximal portion of the aorta. The aortic stenotic murmur is quite constantly attended by a palpable thrill.

The majority of systolic murmurs encountered at the a area are produced not by organic stenosis of the aortic v which is a comparatively rare lesion, but by roughening or tation of the proximal portion of the aorta. The murmur ar from these causes does not possess the initial intensity of aortic stenotic murmur; its quality is distinctly less harsh an musical than that of the stenotic murmur; and it is attende a pure or accentuated second sound at the aortic area.

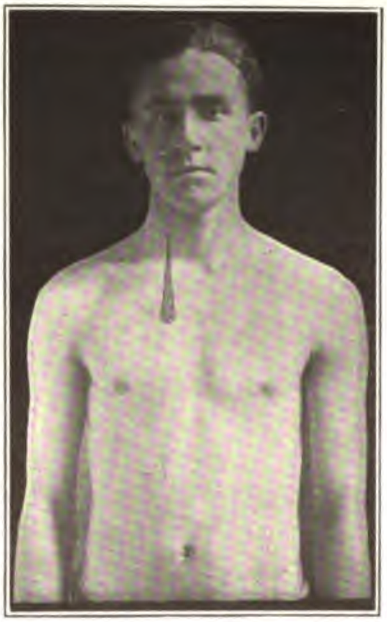


Fig. 150-*A*.—Point of maximum intensity and line of transmission of aortic systolic murmur.

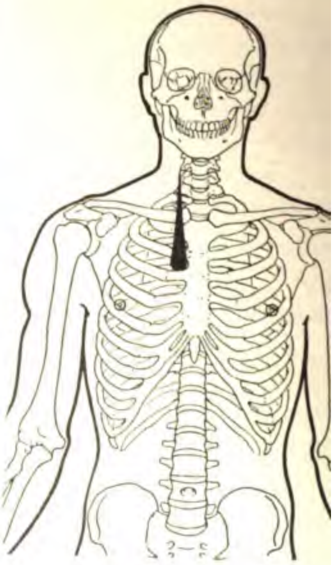


Fig. 150-*B*.—Point of maximum sity and line of transmission of aorti tolic murmur.

Aortic Diastolic Murmur.—A diastolic murmur with its p of maximum intensity at the aortic area is indicative of a regurgitation. The underlying lesion may be shrinking and t ening of the cusps, rupture or perforation of a cusp, or the pres of warty verrucosities upon the cusps, which prevent their a rate coaptation during ventricular diastole. Or again, the mur may arise as the result of the inability of normal cusps to an abnormally large aortic orifice, constituting in this inst *relative aortic regurgitation.*

The aortic diastolic murmur is most clearly audible at aortic area, as a rule, and it is propagated thence in a direc ranging downward and obliquely across the sternum toward the cardiac apex. While the murmur is usually most intense at the aortic area, in certain cases it is to be heard most clearly over the upper portion of the gladiolus just below the Angle of Louis or over the cardiac apex, whence it is transmitted downward and toward the left axillary region. The murmur occurs during diastole, masking or replacing the second sound of the heart at the aortic area. It is loud and blowing, but commonly is not harsh or unmusical as in the case of the aortic stenotic murmur.

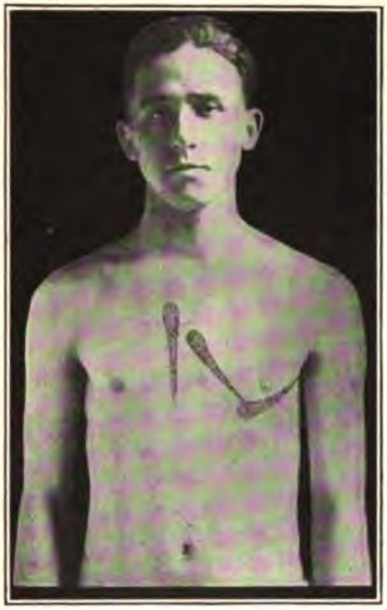


Fig. 151-*A*.—Points of maximum intensity and lines of transmission of aortic diastolic murmur.

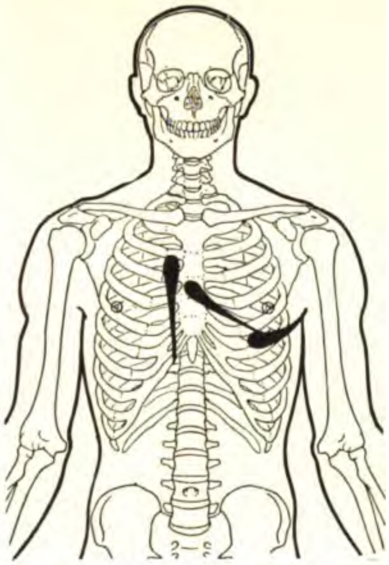


Fig. 151-*B*.—Points of maximum intensity and lines of transmission of aortic diastolic murmur.

While the murmur of aortic regurgitation may occur alone, it is not infrequently accompanied by a systolic aortic murmur, due to coincident stenosis of the valve, a deformity of the aortic valve underlying both conditions, and causing both stenosis and regurgitation. In such event a double murmur is generated at the aortic valve, which is harsh during systole, and less harsh or actually blowing and musical during diastole. Such a double murmur is apt to be confused during a casual examination with a pericardial friction sound with its to-and-fro rhythm, corresponding indefinitely with the systole and diastole of the ventricle.

TRICUSPID MURMURS

Murmurs are not frequently encountered at the tricusp
fice; but when they are present, they are either presystolic
tolic in time, as they indicate stenosis or regurgitation
orifice of the heart.

Tricuspid Presystolic Murmur.—The tricuspid stenotic m
is audible in its maximum intensity in the tricuspid area o
lower portion of the gladiolus, whence it is not transmitt
is due to tricuspid stenosis, which is usually of congenital
The murmur is commonly associated with a thrill over t
cuspid area together with an enfeebled pulmonic sound.
murmur is usually attended by moderate dyspnea, and by
of general venous stasis.

Tricuspid Systolic Murmur.—A systolic murmur arising
tricuspid valve is indicative of tricuspid regurgitation. Th
ative lesion may be a deformity of the cusps of the valve, a
as a sequence of ulcerative endocarditis; but more comm
systolic murmur at this valve indicates *relative tricuspid*
gitation, which is the result of increased blood pressure
pulmonary circulation and engorgement of the right ven
occurring as the result of an obstructive disease of the lu
valvular lesions of the left heart.

The point of maximum intensity of the tricuspid systolic
mur is not as definitely circumscribed to the valve area as
case with murmurs arising at other valves of the heart; but
ful auscultation of the precordia will usually serve to loca
in the tricuspid area, whence it is transmitted by the regur
ing blood stream toward the right and upward.

The first sound of the heart is obscured or replaced b
murmur in the tricuspid area. The pulmonary second sou
ries in intensity with the concomitant state of the myocar
In the presence of simple, isolated tricuspid regurgitatio
second sound is unaltered in intensity or is slightly enfe
whereas in relative tricuspid regurgitation the second sou
accentuated during the maintenance of right ventricular h
trophy, to become enfeebled with the supervention of righ
tricular dilatation.

When relative tricuspid regurgitation develops durin
course of mitral regurgitation, it is instructive to compar
relative intensity of the systolic murmurs occurring at the
and at the tricuspid valves. With failing compensation i

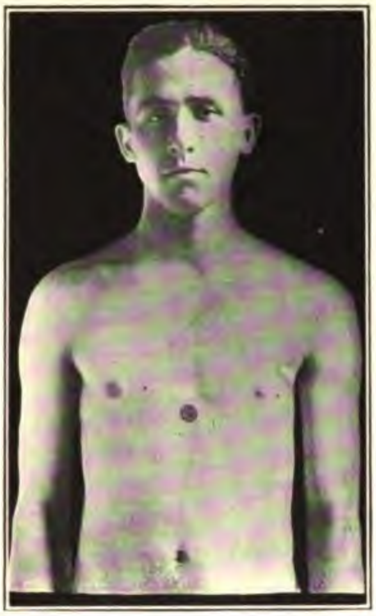


Fig. 152-*A*.—Point of maximum intensity of tricuspid presystolic murmur.

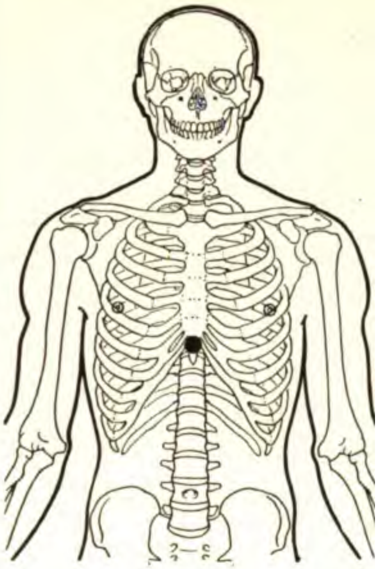


Fig. 152-*B*.—Point of maximum intensi of tricuspid presystolic murmur.

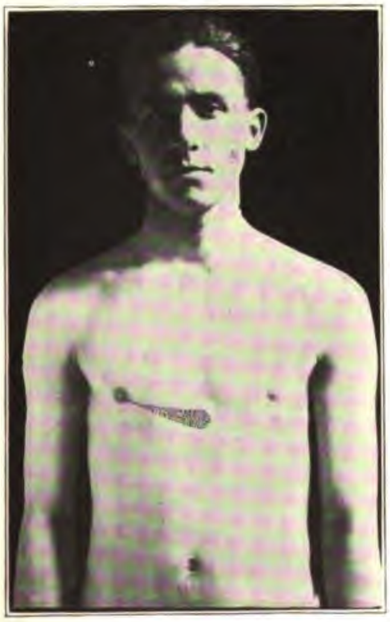


Fig. 153-*A*—Point of maximum intensity and line of transmission of tricuspid systolic murmur.

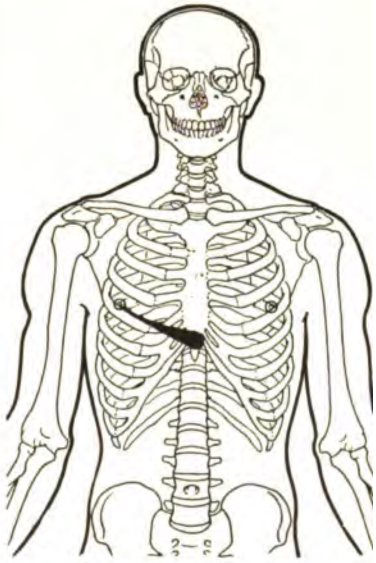


Fig. 153-*B*.—Point of maximum inte sity and line of transmission of tricusp systolic murmur.

be noted that there is a progressive augmentation of the sity of the tricuspid systolic murmur over that of the mitral mur until the latter is masked entirely by the unduly i tricuspid murmur.

The tricuspid systolic murmur is commonly blowing or wh in quality. In the presence of regurgitation of considerable nitude there is to be noted systolic pulsation in the jugular and occasionally systolic pulsation of the hepatic region.

PULMONARY MURMURS

Murmurs at the pulmonary area are of very frequent o rence, but organic disease of this valve is quite rare. The m ity of systolic murmurs encountered at the pulmonary valv functional, and will be discussed in a subsequent parag Murmurs generated at the pulmonary valve are systolic and tolic in time, as they are occasioned by stenosis or regurgit at the valve.

Pulmonary Systolic Murmur.—An organic murmur at the monary valve is occasionally encountered as an evidence o ganic change in the valve. It is usually a sign of pulmo stenosis from a congenital defect. The murmur occurs du ventricular systole; its point of maximum intensity corresp accurately with the pulmonary valve area; it is harsh and u sical when due to organic stenosis of the valve; and it is tr mitted upward toward the root of the neck. The second s of the heart as appreciated at the pulmonary area is impure replaced by a diastolic murmur.

The systolic murmur of *relative pulmonary stenosis,* whic audible in the presence of dilatation of the proximal portio the pulmonary artery, is less harsh than is the pulmonary s otic murmur, and is usually unattended by dyspnea, cyanosi signs of right heart failure. In this case the pulmonary se sound is unimpaired or shows slight accentuation.

The murmur of pulmonary stenosis may be confused duri casual examination with the systolic and diastolic bruit prod by patency of the interventricular septum, *Rogers' murmur.* congenital defect of the heart is attended by a loud and ras murmur with its point of maximum intensity in the fourth intercostal space between the left sternal and midclavicular l The murmur is commonly attended by a palpable thrill. murmur continues throughout systole and diastole, attainin

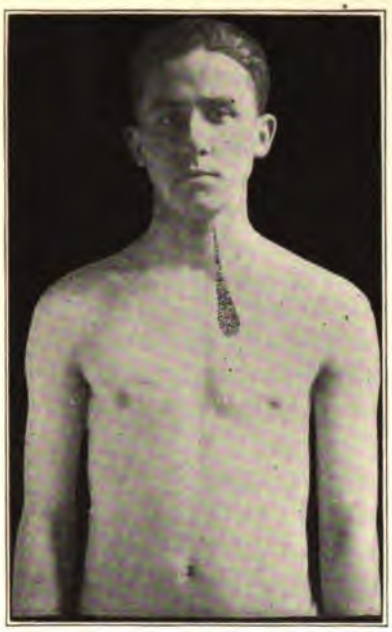


Fig. 154-*A*.—Point of maximum intensity and line of transmission of pulmonary systolic murmur.

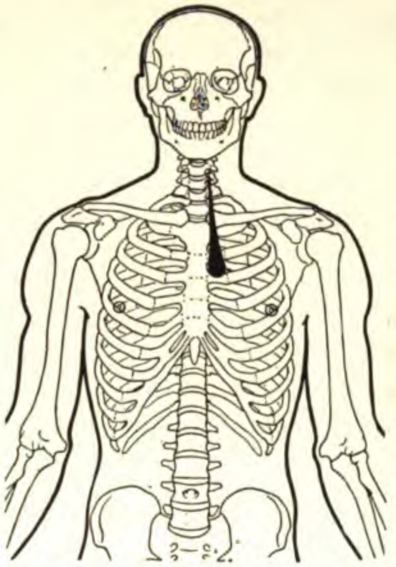


Fig. 154-*B*.—Point of maximum intensity and line of transmission of pulmonary systolic murmur.

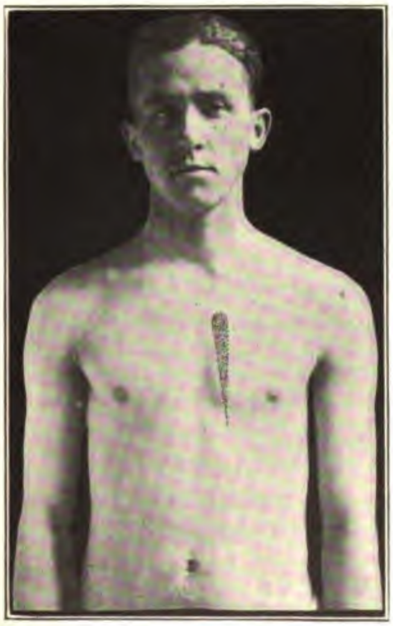


Fig. 155-*A*.—Point of maximum intensity and line of transmission of pulmonary diastolic murmur.

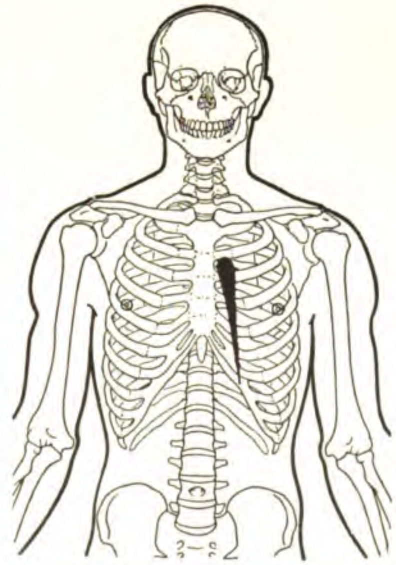


Fig. 155-*B*.—Point of maximum intensity and line of transmission of pulmonary diastolic murmur.

maximum intensity at the height of systole, and exhibiting a progressive diminution of intensity during ventricular diastole.

Moreover, in the presence of fibroid retraction of the anterior border of the left lung, when an extensive area of the right ventricle is exposed directly to the anterior thoracic wall, the conus arteriosus of the right ventricle is likely to suffer compression during ventricular systole, with the consequent production of a systolic rumble in the conus. The murmur arising from the conus is obliterated during forcible inspiration, which causes the anterior pulmonary borders to intervene between the ventricle and the chest wall; and, moreover, the murmur is not attended by signs of embarrassment of the right heart.

Pulmonary Diastolic Murmur.—A diastolic murmur at the pulmonary area is significant of pulmonary regurgitation, due to alteration in the integrity of the cusps of the valve incident to ulcerative endocarditis, or occurring as a result of yielding of the muscular ring of the orifice so that the normal cusps do not close the abnormally large opening, *relative pulmonary insufficiency.*

The point of maximum intensity of the murmur is localized in the pulmonary area in the second left interspace, whence the murmur is propagated downward along the left sternal border. The murmur is audible during ventricular diastole, masking or replacing the second sound at the pulmonary area. In quality the murmur resembles the murmur of aortic regurgitation, save that it lacks the initial intensity of this murmur.

The *Graham-Steel murmur* is a diastolic pulmonary murmur, audible in its maximum intensity in the third left interspace adjacent to the sternum, soft and blowing in quality, replacing the second sound of the heart in this area, and frequently audible only in the recumbent posture. It is significant of stretching of the muscular ring of the pulmonary valve and engorgement of the right ventricle.

FUNCTIONAL MURMURS

Functional murmurs, which are also termed *inorganic, accidental,* and *hemic* murmurs, are endocardial bruits which arise in a heart which is not the seat of permanent structural change. Functional murmurs are not caused by valvular deformity; but they are attributed to excessive fluidity of the blood incident to anemia, to temporary myocardial weakness during the course of acute fevers, or to stretching of the valvular orifices as a result of excessive physical effort.

Functional murmurs are audible most frequently at the pulmonary area, and least frequently at the aortic area. They are more commonly encountered at the mitral than at the tricuspid area.

Functional murmurs are transient, present and absent at consecutive examinations, and do not persist for any considerable length of time. They occur during ventricular systole, but frequently do not consume the entire systole of the ventricle. Functional murmurs are soft, blowing and of low pitch, and are not transmitted beyond the limits of the precordia. When due to anemia they are often accompanied by arterial murmurs or the humming-top murmur in the jugular veins. Moreover, functional murmurs are not attended by changes in the character of the radial pulse, and are not accompanied by appreciable changes in the outline of the heart.

MULTIPLE MURMURS

While organic and functional murmurs arising at the various valves of the heart are separate entities and have been described singly, it is to be borne in mind that two or more murmurs may coexist in the same subject, the differentiation of which is not infrequently attended with considerable difficulty.

When two distinct murmurs are encountered at two valve areas, as for instance at the aortic and the mitral areas, the mere presence of two murmurs at two valves does not necessarily signify organic disease of both valves, as one murmur may be relative. Thus, organic regurgitation at the aortic valve may be attended by dilatation of the left ventricle and yielding of the mitral ring. The mitral murmur in this instance is due to relative mitral regurgitation. Similarly, in organic mitral insufficiency there is not infrequently an associated relative tricuspid regurgitation.

When two murmurs occur at two phases of the cardiac cycle, one systolic and the other diastolic, this fact is of considerable aid in the differential diagnosis. When, however, two murmurs occurring at the same phase of the cardiac cycle are discovered, the differentiation must rest largely upon the points of maximum intensity and the lines of transmission of the murmurs. The quality of the murmurs in this instance is of some assistance, remembering the general rule that stenotic murmurs are harsh and unmusical, while regurgitant murmurs are generally soft, blowing or musical. If, on the contrary, both murmurs are alike in quality, it is probable that there is only one murmur, which is transmitted from the orifice where it is generated to a second valve area, as in the case of the transmission of the murmur of aortic regurgitation to the apical area of the heart.

Moreover, murmurs arising in the heart must be differentiated from a possible cardiorespiratory murmur by directing the patient to suspend respiration, whereupon the latter will disappear. The differential points between endocardial murmurs and the pericardial friction sound are detailed in a subsequent paragraph. Finally, in the differentiation of multiple murmurs the auscultatory findings must be correlated with the general appearance of the patient and such accessory signs as edema, dyspnea and cyanosis.

THE CARDIORESPIRATORY MURMUR

When a segment of the anterior border of the lung is anchored by pleural or pleuropericardial adhesions between the ventricle and the thoracic wall, and when during the course of hypertrophic emphysema, chronic ulcerative phthisis, or massive pleural effusion the anterior borders of the lungs intervene between the heart and chest wall, there is frequently generated a systolic "blow" or "whiff," which is detected near the cardiac apex, along the left border of the heart or even to the right of the sternum, the *cardiorespiratory murmur*. The sound elicited under these circumstances should in all cases be assigned to its real cause; namely, the sudden expulsion of air from a portion of the lung by the impact of the heart during systole. Occasionally the sound is encountered in the absence of pulmonary disease when the left ventricle is excessively hypertrophied.

The intensity of the cardiorespiratory murmur is generally accentuated upon deep inspiration; and the murmur not infrequently becomes inaudible upon forced expiration, as a result of the expiratory recession of the anterior pulmonary borders. Not infrequently the murmur is suppressed when the subject assumes the recumbent posture. While the adventitious sound occurs during ventricular systole, it does not coincide accurately with the commencement or termination of this phase of the cardiac cycle, it is usually audible during the middle of systole.

PERICARDIAL FRICTION

In the presence of inflammation of the pericardium the surfaces of the visceral and parietal membranes, which glide noiselessly over each other during health, become roughened, and a friction sound is generated, the intensity and quality of which depend upon the degree of pericardial inflammation and the power of the cardiac contractions. Depending upon the state of the pericardial surfaces, a sound is produced which varies in quality from that of the sound which is produced upon gently stroking a silken fabric to a loud, rasping sound similar to the sound produced by the creaking of new leather. The pericardial friction sound corresponds roughly with the sounds of the heart, but not with the same accuracy as do endocardial murmurs, as the phases of the friction sound last longer than do the normal heart sounds. To these sounds there may be added a presystolic friction sound occurring during auricular systole.

As a rule, the friction sound is most distinctly audible along the left sternal border between the second and fourth intercostal spaces, though in certain instances it is limited to the region of the cardiac apex.

In differentiating pericardial friction from endocardial murmurs, the examiner should bear in mind the inherent differences between the two phenomena. Endocardial murmurs bear a far more definite relation to the events of the cardiac cycle than do the phases of the pericardial friction sound. Pressure exerted upon the precordia by the bell of the stethoscope frequently serves to accentuate the intensity of pericardial friction, but the same maneuver is without effect upon the intensity of endocardial murmurs.

Forced inspiration exerts quite a different influence upon the sounds emanating from the two sources. Thus, full inspiration by exerting pressure upon the pericardial surfaces intensifies the pericardial friction sound, while the same act causes a diminution in the intensity of endocardial murmurs by the introduction of the anterior borders of the lungs between the heart and the thoracic wall. While many endocardial murmurs are transmitted far beyond the limits of the precordia, pericardial friction is limited to the precordia, and frequently it is audible over only a restricted portion of this area.

Changes of posture exert a more striking influence upon the intensity of pericardial friction than is the case with endocardial murmurs. The friction sound is intensified by bending the trunk forward, and the sound frequently disappears when the subject assumes the recumbent posture. Upon auscultation of the precordia, the impression is conveyed to the examiner that the pericardial friction sound is generated immediately beneath the integument, whereas endocardial murmurs seem rather to come from the depths of the thorax. Moreover, pericardial friction is an evanescent sound; frequently present and absent upon consecutive examinations, and changing in intensity from hour to hour, changes which are not observed in organic murmurs of the heart.

In certain cases the outline of the area of cardiac dullness is of assistance in the differentiation, that of pericarditis differing essentially from that which attends the cardiac enlargement incident to chronic valvular disease.

Pericardial friction must be differentiated from certain substernal sounds of doubtful etiology. In the presence of cardioptosis the cardiac sounds, as elicited over the lower sternal region, frequently possess a harsh scraping or scratching quality, the *xyphosternal crunch*, which may be mistaken for the pericardial friction sound. Of uncertain etiology, this sound is detected in a good many normal subjects.

Pleuropericardial friction, generated in the presence of inflammation of the pleura in relation with the pericardium, is usually to be detected along the left border of the precordia or in the region of the cardiac apex. The sound in this instance bears a definite relation to the movements of the heart; but it is also influenced by the excursions of the anterior pulmonary borders. If the area of inflammation is situated upon the visceral pleura of the left lung, in contact with the pericardium, the sound is abolished during forced expiration, at which time the lappet of lung recedes from the heart. Again, if the area of inflammation is situated upon the costal pleura, anterior to the heart, during forced inspiration the sound is enfeebled or abolished by the intervention of the anterior border of the left lung between the pericardium and the inflamed pleura.

PERICARDIAL SUCCUSSION SOUND

In the presence of air and fluid in the pericardium during the course of hydropneumopericardium, a medley of splashing and gurgling sounds is generated by the movements of the heart, the *pericardial succussion sound*. The sounds in certain cases possess a metallic consonance, hence the name ***metallic gurgle,*** sometimes employed to describe them. In other instances the sound is analogous to that which is produced by a water-wheel in motion, whence the name ***bruit de moulin.*** The sound is occasionally audible without the aid of the stethoscope; and, upon auscultation of the precordia, its intensity may completely obscure the cardiac sounds. The sound is not influenced by the suspension of respiration.

VASCULAR MURMURS

As endocardial murmurs are generated by deformities of the valves of the heart and by alterations in the density of the blood, so also under somewhat similar conditions murmurs are generated in the arteries and veins.

ARTERIAL MURMURS

Auscultation of the arteries may reveal the presence of murmurs in the aorta, the carotid, subclavian, brachial and femoral arteries. During auscultation of peripheral arteries the stethoscope should be applied evenly to the integument but not with sufficient force to diminish the lumen of the vessel, as constriction of the lumen will engender an audible bruit in the normal artery.

The Aorta.—Upon auscultation of the aorta in the left interscapular region a systolic murmur in the vessel is a sign of aneurysm of the aorta. The murmur is accompanied by the concomitant signs of aneurysm; namely, dullness on percussion, frequently a palpable thrill, pulsation of the chest wall in many instances, and diverse pressure symptoms. Frequently there is to be detected a palpable tracheal tug.

The Carotids.—Upon auscultation of the carotid artery the first and second sounds of the heart may sometimes be heard, although the first sound is more frequently inaudible. These sounds are not to be confounded with murmurs, as they are merely the normal sounds of the heart which are transmitted along the course of the blood stream. Endocardial murmurs, however, are similarly transmitted, a harsh systolic murmur audible over the vessel signifying aortic stenosis, aortic roughening or aneurysm of the arch. The transmitted second sound of the heart may be replaced by the diastolic murmur of aortic regurgitation.

The Subclavian Artery.—In certain cases of apical pulmonary tuberculosis a systolic murmur is audible over the course of the subclavian artery. The murmur in this case is due to constriction or to bending of the lumen of the vessel by the traction of pleural adhesions. A diastolic murmur is occasionally audible in the subclavian artery in aortic regurgitation.

The Femoral Artery.—In many cases of aortic regurgitation a double murmur, systolic and diastolic, is to be elicited by auscultation of the femoral artery. The systolic murmur results from the sudden injection of blood into the aorta; while the diastolic murmur results from the reflux of blood during diastole, since at this time the intraventricular pressure is lower than the pressure in the peripheral circulation. This double murmur constitutes *Duroziez's sign* of aortic regurgitation. Friedreich elicited the sign with great constancy in aortic regurgitation, and also found it present with aneurysm of the aorta and in left ventricular hypertrophy with competent aortic valves.

VENOUS MURMURS

A continuous murmur may be elicited over the jugular vein in the normal subject by tightly applying the bell of the stethoscope to the integument over the course of the vessel. A similar murmur may occasionally be elicited by turning the head far to one side, due to constriction of the jugular vein by bands of the cervical fascia and the omohyoid muscle. Hence, during auscultation of this vessel the stethoscope should be lightly applied to the integument, and the head should be maintained in a symmetrical position.

The Venous Hum.—The principal diagnostic sign afforded by auscultation of the venous system is the *venous hum, humming-top murmur, nun's murmur*, or *bruit de diable*. This murmur which is continuous, has been compared to the sound of the buzzing of insects and to the sound of a circular saw in action, comparisons which give but an imperfect conception of the quality of the sound.

The sound is commonly to be elicited over both jugulars, but it is more intense, as a rule, over the right vein. The murmur has its maximum intensity over the inner third of the clavicle. The intensity is increased when the patient assumes the upright posture, during inspiration, and during diastole, factors which favor a more rapid flow of blood through the veins.

The murmur is generated in the bulb of the jugular vein and not in the carotid artery, as described by Laennec. The intravenous origin of the murmur is demonstrated by the facts that the murmur is continuous and is not intermittent as are arterial bruits; and that it can be entirely suppressed by light compression of the jugular vein, a compression too insignificant to appreciably influence the action of the carotid circulation.

The venous hum is occasionally to be elicited in the young subject during health; but in the vast majority of cases its presence points to anemia, especially chlorosis and pernicious anemia.

Friedreich has detected a similar venous murmur in the right interscapular region at the level of the third and fourth dorsal vertebrae, which occurred in conjunction with the venous hum in the jugular veins, and which he attributed to excessive fluidity of the venous content of the superior vena cava.

CHAPTER XVI

SPHYGMOMANOMETRY

Blood pressure may be estimated by several types of sphygmomanometer, the procedure constituting sphygmomanometry. For practical work one of the modifications of the Riva-Rocci instrument will be found generally satisfactory, the basis of all sphygmomanometers of this type consisting of an inflatable rubber bag contained in an inelastic cuff, a mercury manometer, and an air pump so connected by rubber tubing that the air which is pumped is distributed with equal pressure to the cuff and the manometer. A further type of instrument, which dispenses with the use of the mercury manometer, and which instead of recording the pressure in millimeters of mercury records the pressure upon a dial, is the Rogers Tycos sphygmomanometer.

With either type of instrument, in recording the blood pressure two methods may be employed; namely, the palpatory method, and the auscultatory method. Whichever method is employed, certain details of the technic must be observed in order to obtain accurate results. The pressure is usually recorded from the brachial artery during the routine examination, as it is readily accessible and is almost on a level with the heart. The cuff should be applied to the arm at least two inches above the bend of the elbow; the connections of the tubing to the different portions of the instrument should be air-tight; the dilatable rubber bag should be carefully adapted to the inner aspect of the arm, overlying the brachial artery; and the cuff should be snugly applied, but not with sufficient force to interfere with the venous return. No fabric should be permitted to intervene between the cuff and the integument; and the clothing of the extremity should be removed in order to avoid constriction of the artery by rolling up a sleeve.

Clinically it is desirable to estimate the blood pressure during ventricular systole (systolic pressure); during ventricular diastole (diastolic pressure); and to determine the difference between these figures (pulse pressure).

Palpatory Method.—With the instrument properly adjusted to the arm, air is slowly pumped into the cuff and manometer until the pulse becomes inappreciable to the fingers palpating the radial artery. When the pulse disappears, the mercury is pumped up 10 or 15 millimeters above this point; and the release valve is turned, allowing the mercury column to slowly descend. At the instant that the pulse becomes again appreciable at the wrist the release valve is closed, and the *systolic pressure* is read upon the scale or dial.

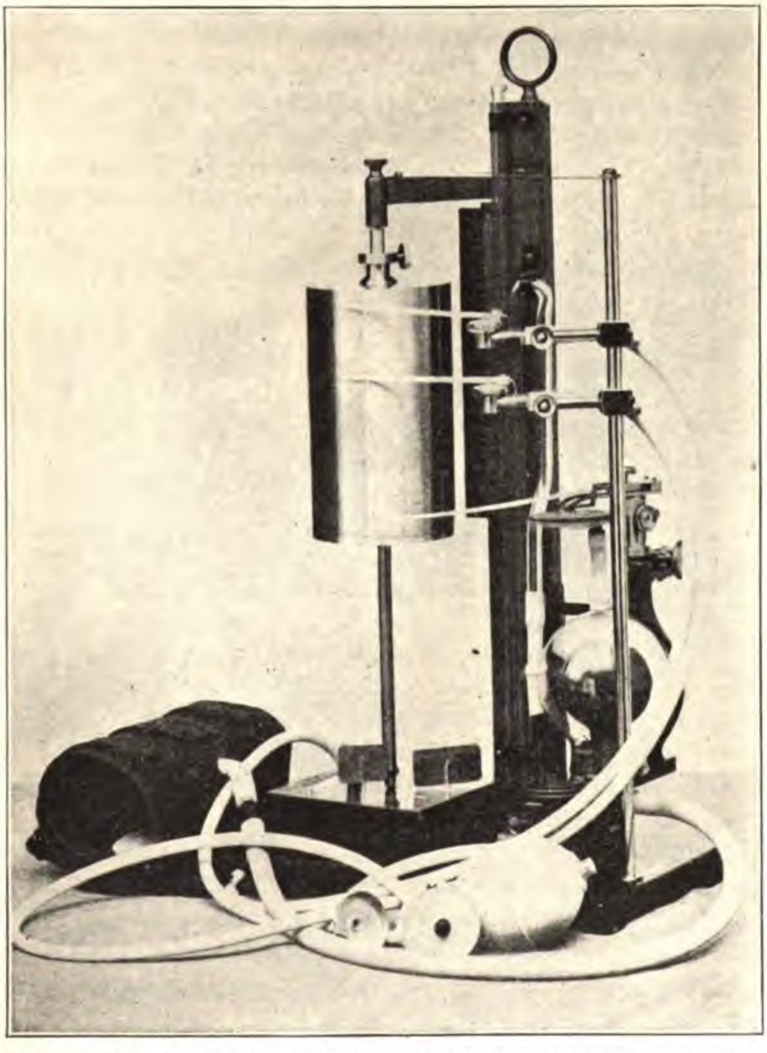


Fig. 156.—The Erlanger sphygmomanometer with the Hirschfelder attachments by means of which simultaneous tracings can be obtained from the brachial, carotid, and venous pulses. (From Warfield.)

The principle involved in this procedure is that it requires an amount of external pressure to obliterate the pulse in the artery which is commensurate with the intravascular pressure during systole. It is doubtless true that a certain degree of the external pressure is consumed in overcoming the resistance of the soft tissues, the arterial wall and the blood velocity; but in the normal adult subject this difference is estimated at 7 to 10 millimeters of mercury. By the use of a broad cuff, errors in this direction are reduced to the minimum.

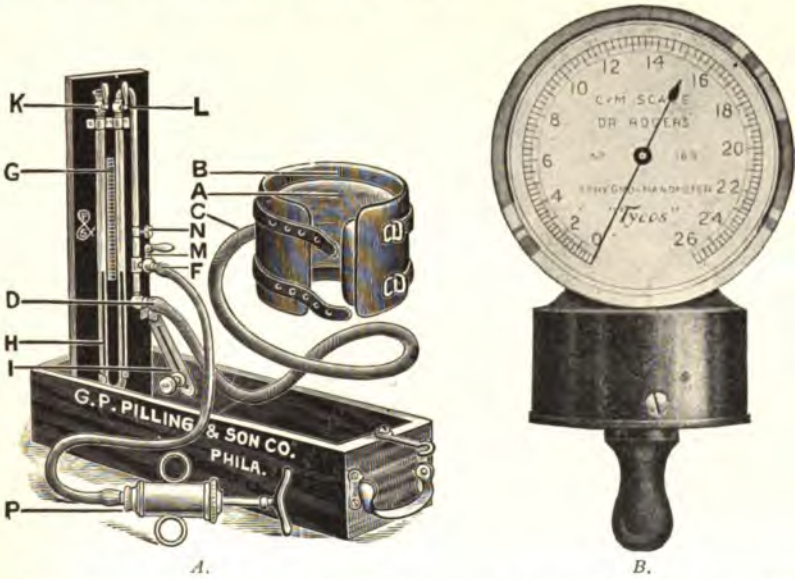
Having determined the systolic pressure by the method described, the release valve is again slowly rotated and the mercury column allowed to descend upon the scale, the undulations of the column being closely observed. The descent is attended by rhythmic oscillations, and at one point in the descent these oscillations become very pronounced, this point corresponding to the diastolic pressure, and as a rule being accompanied by a larger pulse wave than normal at the wrist.

Fig. [illegible]. [illegible] [illegible] use of sphygmomanometer. (From Warfield.)

By deducting the diastolic pressure from the systolic pressure, the pulse pressure is obtained. In the normal adult male subject the systolic blood pressure averages 130 millimeters. The diastolic pressure averages 90 millimeters in the normal adult, representing approximately 70 per cent of the systolic pressure in the normal subject. The pulse pressure in the normal adult varies from 30 to 50 millimeters, representing approximately 15 to 20 per cent of the diastolic pressure. However, a pulse pressure of 60 millimeters represents the extreme limit of safety in the normal subject; and a pulse pressure above this figure is to be considered distinctly pathological.

Auscultatory Method.—The auscultatory method of estimating the blood pressure is more accurate than is the palpatory method; and, in addition, it shows wider ranges of pressure in the individual case. The systolic pressure, as determined by the auscultatory method, is always from 7 to 10 millimeters above that registered by the palpatory method, whereas the diastolic readings range from 10 to 15 millimeters below those obtained by the palpatory method.

In estimating blood pressure by this method the bell of the stethoscope is applied over the brachial artery just above the bend of the elbow, and the cuff is inflated until all sound disappears. Having attained this point, the air is allowed to slowly escape from the cuff, whereupon a series of sounds are audible, which have been divided into five phases.



A. *B.*

Fig. 158.—*A*. The Faught blood pressure instrument. An excellent instrument which is quite easily carried about and is not easily broken. (From Warfield.) *B*. Detail of th dial in the "Tycos" instrument. (From Warfield.)

The *first phase* is represented by the first sound which is audible, and represents the proper point at which to record the *systolic pressure*. The sound is attributed by Erlanger to the water-hammer action of the blood column at the instant of its contact with the relatively stationary column below the point of constriction. The first phase is quickly followed by a peculiar murmuring sound as the tension in the cuff is lowered, the *second phase*, which is attributed to the induction of "fluid veins" in the blood the age of the subject; but with advancing age in the adu ject there is a progressive increase in blood pressure.

Pathologic Variations.—When a high *systolic pressure* countered in a patient who is and has been in repose, it poi cardiac hypertrophy, the causes of which are to be soug chronic nephritis, whether a glomerulonephritis or a diffus rosis of the kidney; to arteriosclerosis, or increased intrac pressure. In the pregnant woman it may point to threa eclampsia. In cardiovascular disease a high systolic pr which is accompanied by an increased pulse pressure usua

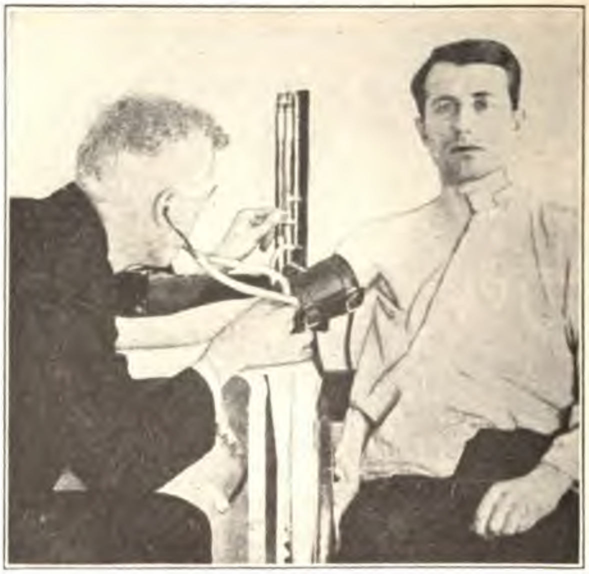


Fig. 161.—Observation by the auscultatory method and a mercury instrument. On regulates the stopcock which releases air gradually. (From Warfield.)

indicative of adequate compensation; whereas a normal or ered systolic pressure with a diminished pulse pressure poin threatened cardiac failure.

In the presence of autointoxication the systolic pressu frequently elevated as the result of the elaboration of toxic in the gastrointestinal tract.

A decrease in the systolic pressure accompanies extensive of blood, internal or external, malnutrition, the cachexia o lignant disease, chronic alcoholism, myocardial degenera pericardial effusion, orthostatic albuminuria, and the vas asthenia of Addison's disease.

The importance of variations in the *diastolic pressure* has come to be generally recognized. The diastolic pressure represents the degree of peripheral resistance which must be overcome before the left ventricle may discharge its contents; and if this pressure is raised, as it is in arterial fibrosis and chronic renal inflammation, an added burden is thrown upon the heart before the blood can begin to circulate. Moreover, when a high diastolic pressure is encountered, it is of the first importance from the standpoint of prognosis and treatment to determine whether it is accompanied by an increase in the systolic pressure, or whether the increase is due to functional angiospasm. In aortic regurgitation the diastolic pressure is low, while the systolic pressure is commonly increased, giving a very large pulse pressure. In toxic goitre the diastolic pressure is likewise lowered, with an approximately normal systolic pressure.

The *pulse pressure* deserves careful study in all cases in which sphygmomanometry is practiced. The pulse pressure represents the contractile power of the left ventricle in excess of the diastolic pressure; or, in other words, the power of the left ventricle over and above the peripheral resistance. During the course of lobar pneumonia, when the heart is laboring under an increased load, a daily record of the pulse pressure gives valuable information as to the state of the myocardium, and affords a valuable prognostic and therapeutic index. As the overtaxed heart fails, the systolic pressure gradually approximates the diastolic pressure, the pulse pressure steadily diminishing until the point is reached at which the coronary circulation is inhibited and the nuclear centers are no longer supplied with blood, the point at which life becomes impossible. A systolic pressure of 60 millimeters seems to be the minimum pressure compatible with life, although lower pressures are recorded in the earlier literature.

Venous Pressure.—Hooker, who has devised an apparatus which permits the recording of the venous pressure, finds a progressive rise of venous pressure from youth until old age. Impairment of the myocardium, attended by lowering of the systolic arterial pressure, is accompanied by a concomitant rise of venous pressure. Frequently an increase in venous pressure is the earliest sign of failing compensation. Clark finds that a venous pressure of 20 centimeters of water represents the limit between adequate compensation and decompensation of the heart, and that a rise above this point is apt to be followed by cardiac failure.

A rough estimate of the venous pressure may be made by observing the superficial veins upon the dorsum of the hand when the latter is raised above the level of the heart. Normally these veins should collapse when the hand is raised above the level of the heart, and they should practically collapse with the hand at the level of the cardiac apex. But in the presence of increased venous pressure the veins fail to collapse when the hand is raised even above the level of the heart.

Oliver states that the venous pressure may be estimated in millimeters of mercury by multiplying by 2 each inch above the level of the cardiac apex at which the veins collapse.

CHAPTER XVII

THE DIAGNOSIS OF ABNORMALITIES OF THE HEART BEAT

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INTRODUCTION

For an observer to be able to recognize and interpret correctly abnormal signs that may occur during the examination of an organ, he must be conversant with the evidences of its normal action, and the causes of their manifestation. Before entering upon a discussion, therefore, of the abnormalities of the heart beat and their causes, a brief review will be made of the factors which underlie the production and regulation of the normal heart beat. Consideration will be given also to a description of the physical signs which accompany the normally beating heart and the manner of their production clinically. Further than this, certain factors necessary to normal cardiac function will be reviewed which give no direct clinical evidence of their interplay, but whose presence can be demonstrated, and whose function must be understood before one attempts to interpret variations from the normal.

The Signs of Normal Heart Action.—The signs which give evidence of the beating of the normal heart are: (1) an impulse against the chest wall, (2) a double sound with each cycle and (3) a pulse wave in the peripheral arteries. These are all, broadly speaking, phenomena of ventricular origin. It is only by indirect evidence that the clinician is aware of the action of the normal auricle. The ventricle* constitutes the main bulk of cardiac musculature; it is the pump whose continued working is essential to the life of the organism. The auricle receives the blood, but the ventricle must send it to lungs and periphery. Evidences of disease elsewhere in the heart are of importance only as they may indicate an interference, direct or indirect, with ventricular function.

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*In what follows, the term ventricle will be used to refer to the musculature of both ventricular chambers.

This supreme importance of the ventricle and the fact that it alone gives clinical evidences of its action, result in its being ordinarily thought of not as a part of the heart, but as the heart itself. And so one speaks of systole and of diastole without taking the trouble to say that he means to refer not to auricular but to ventricular phases. Such expressions as "weak heart action," and "irregular heart action" have reference, strictly speaking, only to the action of the ventricle. The auricles, indeed, may be regarded as accessories, the ventricular musculature as *the heart*.

But it is within this accessory part of the normally beating heart that the rate and observed ventricular rhythm arise and are controlled; and it is, broadly speaking, due to disturbance there (or to the assumption by other parts of the heart of functions that normally reside in the auricle) that abnormal ventricular rates or rhythms arise, and are observed clinically.

The Normal Heart Beat

Before describing the manner in which abnormalities of the heart beat are produced by disturbance in the auricular musculature or elsewhere, a brief description will be offered of the manner in which the normal heart beat is elaborated and distributed to the ventricle and of the part played by the auricle in originating and controlling it.

The systole of the heart by which the ventricle empties itself, and which is evidenced clinically by an impulse against the chest wall, by the first heart sound and by the pulse, is not a simultaneous contraction of the whole heart musculature, but is an orderly contraction which begins at a definite area, spreads in an orderly fashion and involves different muscle parts in sequence.

Rhythmicity.—One of the inherent properties of heart muscle is *rhythmicity*. By virtue of this property any part of the musculature, even when isolated, under proper conditions, exhibits contractions which recur at regular intervals. This rhythmicity is of higher degree in the auricle than in the ventricle, though it is present in the latter in sufficient degree to cause contractions of that chamber at a slow rate when it is cut off from auricular influence. Rhythmicity in the auricle is highest near the mouths of the great veins and diminishes in degree toward the ventricle.

The Pacemaker.—The area of highest rhythmicity lies at the junction of the superior vena cava and the right auricle. In this area is a specialized mass of tissue, the *sinoauricular node*, in which the beat originates. As the wave* spreads from this area over the auricle, it sets off contraction of successive portions of musculature in turn, and progresses toward the ventricle. These lower portions of the auricular musculature, although inherently rhythmic, possess a lesser degree of rhythmicity than the parts above them, and do not contract until excited by the spreading wave. Thus it is that the upper portion, at the sinoauricular node, possessing the highest rhythmicity, sets the pace for the whole heart, and is called the pacemaker.

Cardiac musculature possesses the property of *conductivity* and by virtue of this attribute the impulse to contraction is conducted not only over the auricle but to the ventricle as well. But it is conducted to the ventricle along a special path.

The Auriculoventricular Bundle.—At the lower edge of the auricular musculature and at the base of the auricular septum (posterior right border) lies another mass of specialized tissue called the *auriculoventricular node*. Beginning at the node, a specialized bundle of tissue, the *auriculoventricular bundle*, runs downward to the membranous part of the ventricular septum. Here it divides into a right and left branch, one upon each side of the septum. Subdivisions of the branches are continued in the endocardium as the Purkinje cells, thus bringing the auriculoventricular bundle into contact with the musculature of the ventricle.

After the impulse has initiated the orderly contraction of the auricle, it is conducted to the musculature of the ventricle along this path. It reaches the various portions of the ventricle at about the same time and causes a contraction of the parts of this chamber which is almost simultaneous for all its parts.

It will be noted that, although the ventricle is inherently rhythmic, its rhythmicity is not of sufficiently high degree to cause it to contract at as rapid a rate as that of the impulses which come down to it from the auricle. The rate of the ventricle, therefore, is dependent upon that of the auricle, and the rate of the latter is dependent upon the area of highest rhythmicity, i. e., the pacemaker.

Summary.—The normal contraction of the heart consists of a contraction of the auricle, followed by a contraction of the ventricle. The beat is initiated at the pacemaker, spreads over auricle, is conducted to the ventricle along the auriculoventricular bundle, and sets up a contraction of the latter chamber. A certain time elapses between the beginning of auricular contraction and the beginning of contraction of the ventricle. This is called the *conduction time*.

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*No attempt is made here to enter into a distinction between the excitation wave and the wave of contraction. For a consideration of these and other more technical matters, the reader is referred to special works on these subjects.

Influence of the Vagi.—After an orderly beat, the heart rests until another impulse is elaborated at the pacemaker, when the cycle is repeated. The time interval between successive beats determines the rate. Now the inherent rhythmicity at the pacemaker is greater than the normal rate would indicate, and tends to drive the heart at a rate faster than the observed average of 72 per minute. But the influence of the vagus nerves restrains, to some extent, the rhythmicity of the pacemaker, and the latter, instead of sending out impulses at a rate of about 150 per minute, is held in check, and releases the impulses at a rate only about half as high. Stimulation of the vagus, by increasing its effect at the pacemaker, slows the rate; diminution of vagus tone allows the pacemaker to assert itself and increase the rate. The degree of vagus tone, then, acting at the pacemaker determines the normal rate.

The vagus affects not only the rate, by its action at the pacemaker, but it influences conduction also. Vagus stimulation depresses the conductivity of the auriculoventricular bundle and thus increases the time interval required for the passage of the impulse from auricle to ventricle.

Graphic Registration of Events in the Cardiac Cycle

Graphic methods of registering certain phenomena associated with the heart beat have made possible more exact knowledge of the mechanism of origin and propagation of the impulse. The electrocardiograph affords the most exact means of elucidating and studying not only the normal but abnormal beats as well. In the following pages, therefore, characteristic electrocardiograms are exhibited, which aid in understanding the normal contraction. With these as a basis for comparison, electrocardiograms which portray characteristic abnormalities of contraction are shown as a means of illustrating graphically the abnormal mechanism involved in the production of the abnormal beats.

Principle of The Electrocardiograph

When a human subject is placed in the circuit of a string galvanometer, each contraction of the heart produces certain characteristic movements of the string. By projecting the shadow of the string upon a moving photographic film, a photograph of the movements of the shadow is obtained. This photographic record is an electrocardiogram. The machine is equipped with a time marker and the completed record shows in graphic form certain events of the cardiac cycle with the time relationship of their occurrence.

Curves from normal subjects show minor differences and no two are exactly alike, but the general form of normal human electrocardiograms is constant and shows certain characteristic waves. These are usually lettered after the terminology of Einthoven which is employed in the accompanying figures.

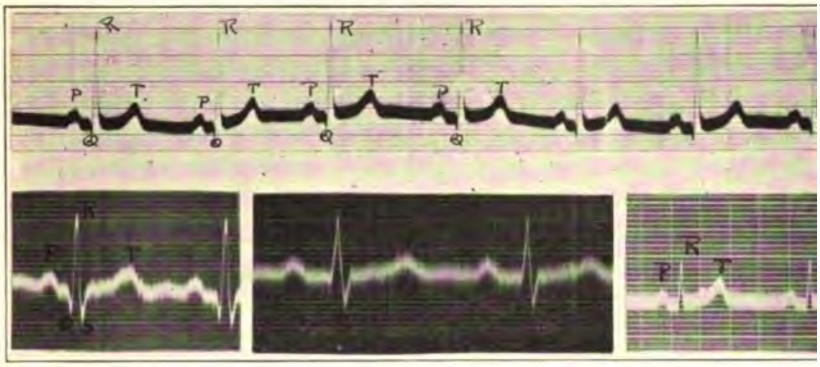


Fig. 162.—Four different specimens of normal electrocardiograms. Lead II. *P*, *R*, and *T* waves occur in each. Both a *Q* and an *S* wave occur in one. In others, one or both of the *Q* and *S* waves are absent.

In Fig. 162, four specimens of normal electrocardiograms are shown. Two broad waves lettered *P* and *T* and a sharp deflection lettered *R* occur in all four specimens. Two smaller waves lettered *Q* and *S* occur in one specimen, while in the other curves one or both of these two waves are absent. Experimental evidence proves that *P* is associated with auricular activity, while the *Q R S T* group is produced by activity of the ventricle. The conduction time, or the *P-R* time as it is often called, is the time, (measured in hundredths of a second) between the beginning of the *P* wave which represents the onset of auricular activity, and the beginning of the *Q* or *R* wave, which portrays the arrival of the impulse at the ventricle.

In such a record, then, is pictured with each systole, a graphic expression of the beginning of auricular contraction at the pacemaker, its spread over the auricle, its transmission to the ventricle and the muscular activity of the latter chamber.

Summary of Introduction

In the preceding pages attention has been called at some length to the manner in which the normal heart beat arises at the pacemaker, spreads over auricle, passes to and involves the ventricle. The graphic registration of these processes has been considered. The physical signs which they produce have been reviewed. In the pages which follow, attention will be directed to abnormalities of the heart beat and the abnormal physical signs which accompany them. This will include due consideration of the abnormal mechanism involved in their production. Reference will be made in each case to characteristic electrocardiograms which graphically portray this abnormal mechanism and aid in its understanding. But since the most of the abnormalities of the heart beat that are observed clinically can also, as a usual thing, be diagnosed at the bedside, without the aid of electrocardiography, reference will be made to the latter only as a means of illustration. Chief consideration will be given to physical signs (and symptoms) which accompany the abnormal mechanism under discussion, and which are sufficient, in most instances, to differentiate it from other abnormalities of the heart beat.

SINUS ARRHYTHMIA

The impulses to contraction, which are elaborated at the pacemaker are, as a rule, approximately rhythmic. Were it not for the restraining influence of the vagus at the sinus area, these impulses would initiate contractions at a rapid rate. The degree of vagus tone at the pacemaker determines the length of the time interval between beats, thus establishing the rate. Now many influences operate to cause changes in the degree of vagus tone, and these changes in tone are reflected in slight variations in heart rate from moment to moment. The beats, therefore, are not perfectly rhythmic, exact measurements showing slight variations in the time interval between them. The resulting arrhythmia, however, is of so slight extent that, as a usual thing, it cannot be detected by feeling the pulse and examining the apex, but requires instrumental means of demonstration.

In certain individuals, however, these variations in rhythm are of such extent that they may be readily noted. Since the arrhythmia in such cases arises at the sinus area it is called ***sinus arrhythmia.*** Fig. 163 is an electrocardiogram illustrative of this condition. The varying interval between auricular as well as between ventricular complexes is apparent. The beats arise at the normal area, spread over the auricle in normal fashion, are conducted to and involve the ventricle. Each complex is normal. The electrocardiogram shows that the whole heart is involved.

The diagnosis of this condition can usually be made without resorting to instrumental means. The important point is to demonstrate vagus influence on the arrhythmia. In many cases this demonstration goes on with each respiration: there is a quickening of the pulse with inspiration and a subsequent slowing as expiration takes place. Such a variation of rhythm with normal respiration is common in young children and is observed not infrequently in individuals at the age of puberty. It is less common in young adults and rare in older subjects.

While not all examples of sinus arrhythmia show a variation synchronous with normal respiration, yet deep breathing may be shown to influence them, thus establishing their vagal origin. They are abolished when the rate is made rapid, as by exercise, and are more pronounced when the rate becomes slow again during rest from exercise. The apex shows the same variation in rhythm as the pulse and is of the same rate as the latter. There is no great difference in the loudness or force of individual beats.

PREMATURE CONTRACTIONS

All portions of heart muscle are inherently rhythmic but only one area, that at the pacemaker, under normal conditions initiates contractions. The electrocardiographic record of contractions which arise at the pacemaker has a characetristic appearance. (See Fig. 162.)

Under various abnormal conditions, however, another area may reaches the hyperirritable focus. Such a focus outside of the impulse from the pacemaker, may itself form an impulse to contraction before the rhythmic impulse from the sinus area reaches the hyperirritable focus. Such a focus outside of the pacemaker is called an *ectopic* focus and the beat which has its origin in an ectopic focus is called an *ectopic beat.* If the ectopic focus is in the auricle, the beat which it initiates is called an *ectopic auricular beat;* if in the ventricle, the beat is called an

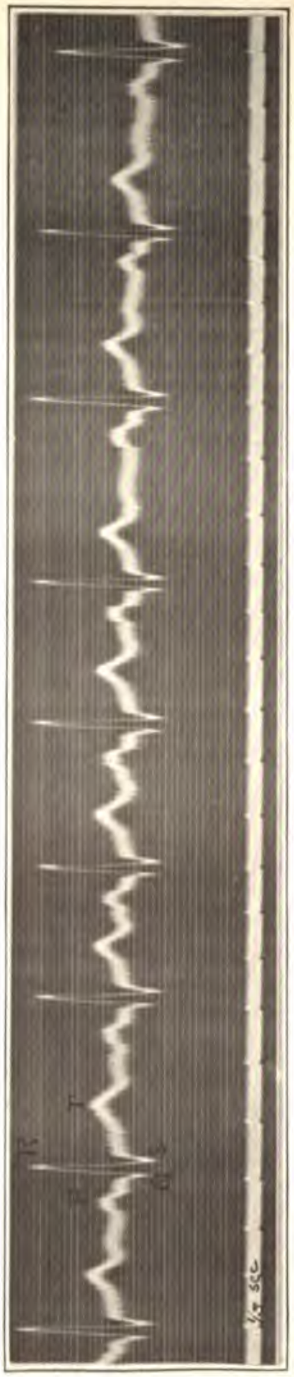
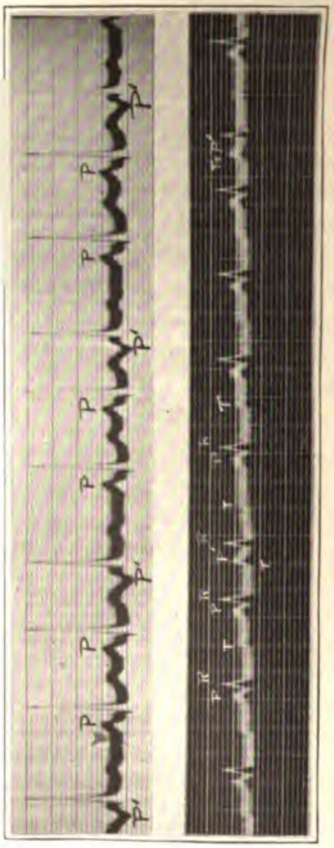


Fig. 163.—Electrocardiogram of sinus arrhythmia. Time in fifths of a second. Note the different time interval between cycles.



ectopic ventricular beat. Since an ectopic beat interrupts the normal rhythm by occurring prematurely, i. e., before its rhythmic period, it is called a *premature contraction.* Fig. 164 shows two electrocardiograms which record premature beats. The ectopic focus from which these beats arose was in the auricle, and contractions of the auricle took place in response to stimuli which originated in the ectopic auricular focus. These auricular contractions produced waves (*P′*) of different appearance from those (*P*) produced rhythmically at the pacemaker. Both the normal and the premature auricular beats are followed by ventricular complexes. The ventricular complexes which follow the premature auricular beats are quite similar in form to those which follow the normal auricular beats. In both cases the impulse reaches the ventricle in much the same way and produces similar ventricular responses.

After the premature beat there is a pause, the interval between the premature beat of the auricle and the succeeding normal beat being longer than the normal interauricular interval. This, however, is not a true compensatory pause. That is to say that the length of this long interval is not quite sufficient, when added to the preceding short interval (between normal auricular beat and premature beat of the auricle) to equal two normal interauricular intervals.

Fig. 165 is an electrocardiogram which records premature beats of different type. The stimuli which produced these abnormal beats arose in the ventricle. Ventricular complexes are recorded, therefore, with no preceding auricular wave. These ventricular complexes are of different appearance from those which follow impulses received from the auricle. After the premature beat there is a ventricular pause, following which the next beat is of normal origin. A contraction of the auricle occurred before the premature ventricular systole was completed. The *P* wave representing this auricular contraction is superimposed on the abnormal ventricular complex and is, for that reason, less readily recognized than the normal auricular waves. This auricular contraction, occurring during the refractory phase of the ventricle, did not produce a contraction of the latter. The next auricular beat, however, is followed by a ventricular beat in the usual way. The pause, therefore, following the premature ventricular beat is a true *compensatory pause.* Its length, being determined by the next sequential auricular beat, is exactly suffi-

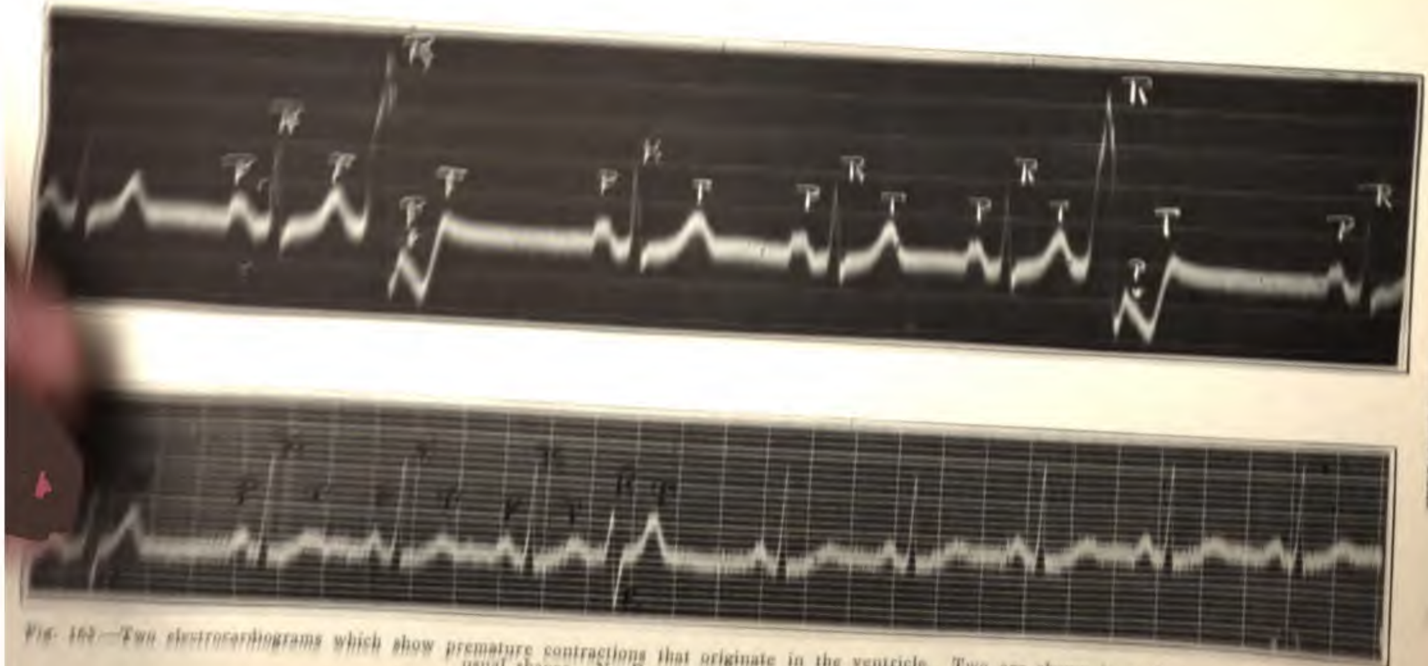


Fig. 162.—Two electrocardiograms which show premature contractions that originate in the ventricle. Two are shown in each record. Note their unusual shapes. No *P* wave precedes the ectopic beats.

cient, when added to the preceding short interval, to equal two interauricular intervals.

It will be noted in both figures that the premature contraction, followed as it is by a pause which is approximately compensatory in the one case and exactly so in the other, does not constitute a true "extra systole," though it is frequently so designated. It interrupts but does not essentially change the rhythm, while the number of beats has not been increased.

It does occasionally happen that premature ventricular contractions occur so early in diastole that the succeeding auricular beat finds the ventricle recovered from its refractory phase and ready to respond. In such a case there is no compensatory pause and an "extra beat" occurs. Such cases, however, are relatively quite rare.

By keeping in mind the mechanism of their occurrence the recognition of premature contractions usually gives no difficulty. The premature contraction, occurring as it does before the ventricle has rested as long as its customary interval, usually gives evidence of its immaturity. It may not be of sufficient force to cause a peripheral pulse; indeed it may not be strong enough to open the outlet valves of the ventricle. The peripheral pulse, when it does occur, is usually smaller as well as earlier than normal.

If the beat is not forceful enough to produce a radial response, there occurs a pulse intermission while signs of ventricular activity are present at the apex. The character of the signs produced at the apex by a premature beat depends upon the force of the premature contraction. If the strength of this is insufficient to open the valves at the base, only the first heart sound is heard accompanying the premature beat. On listening at the apex, then, one hears the early occurrence of one or both heart sounds followed by a long pause. The finger on the radial artery will detect no pulse wave consequent on a single heart sound, and may find none immediately following the premature double sound.

Another evidence of immaturity on the part of premature contractions may be noted in connection with their relationship to existing murmurs. A systolic murmur at the apex is usually less intense with the premature beat, or absent altogether. The occurrence with premature beats of existing aortic murmurs will depend upon whether and to what extent the aortic valves are opened by the premature beat.

Premature beats usually disappear if the heart rate is increased to as much as 100, and are rare at rates higher than 120.

The subject not unusually is conscious of the beat that follows the pause which may produce a "thumping" in the chest.

While careful examination of apex and pulse usually suffices to establish the character of the arrhythmia when premature contractions are present, the differentiation of auricular from ventricular premature beats is usually not certain without instrumental means. An electrocardiogram, however, not only gives information upon this point, but localizes the place of origin still more precisely. In this way premature contractions arising in upper or lower auricle, in right or left ventricle, and in the tissues which join auricle to ventricle, can be differentiated. Certain observations, however, suggest their ventricular origin. These are: (a) a pause following the premature beat which appears to be exactly compensatory rather than slightly less than this; (b) a pronounced wave in the neck veins at the time of occurrence of the premature beat. This is accounted for by the simultaneous occurrence of the premature ventricular and the rhythmic auricular beats. (See Fig. 165.)

HEART BLOCK

The conduction of the impulse from auricle to ventricle requires a certain time. This time interval, called the a-v time, or the *P-R* interval, varies normally within limits which are usually placed at 0.11 or 0.12 to 0.18 or 0.19 of a second. The conducting bundle may be the seat of disease which interferes with the normal transmission of the auricular impulse to the ventricle. The impulses may be delayed beyond the normal time limit of 0.18 of a second, or the interference to their passage may be so great as to prevent altogether their reaching the ventricle. In other cases the impaired conductivity of the auriculoventricular bundle may be of such nature that some impulses fail to reach the ventricle, while others are conducted to it, but with perhaps a longer time interval than normal.

In Fig. 166 some of the auricular waves are followed by ventricular complexes, while others produce no response of the ventricle. In the case of those auricular impulses which succeed in reaching the ventricle the *P-R* time is prolonged. The auricular waves (*P*) occur regularly, the ventricular complexes do not. Examination of the heart, while this record was being obtained,

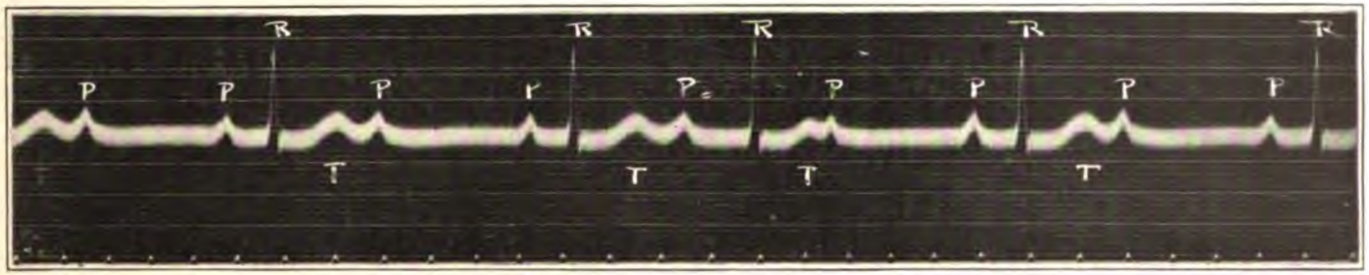


Fig. 166.—Electrocardiogram of partial heart block. Time in fifths of a second. Some *P* waves are followed by ventricular complexes after a lengthened *P-R* interval. Other *P* waves are blocked.

would have shown that the ventricle was beating irregularly, even though the record shows that the auricle was all the time contracting at rhythmic intervals.

Fig. 167 is an electrocardiogram which records events of a somewhat different character. In it, too, the *P* waves occur regularly, but the ventricular complexes are rhythmic also, though occurring at a rate much slower than that of the auricular waves. There is no sustained relationship between the occurrence of auricular and ventricular waves. The latter do not take place because of any influence of the former, as is normally the case. The auricle is beating regularly in response to rhythmic impulses from the pacemaker, while the ventricle, receiving no impulses from above, is beating in consequence of its own inherent rhythmicity, having established a rhythm of its own. Examination of the heart at this time would have shown a regular beating of the ventricle at a rate much slower than that of the recorded auricular contractions, which were occurring at a normal rate.

In the one case (Fig. 166) the conduction of impulses from auricle to ventricle was interfered with. Some of the auricular impulses reached the ventricle in spite of this interference, but after considerable delay. When they did reach it they stimulated the ventricle to contract. Other impulses from the auricle were unable to reach the ventricle, and the latter waited for the next rhythmic auricular stimulus.

In the other case (Fig. 167), the passage of impulses over the conducting system from auricle to ventricle, instead of being somewhat interfered with, was completely blocked. The ventricle, therefore, deprived of auricular stimuli, beat in consequence of its inherent rhythmicity. The former is an example of *incomplete* or *partial heart block*, the latter of *complete heart block*.

In those cases in which the interference to conduction is not great enough to actually block any auricular beats, the condition is called *delayed conduction*. Such a condition is portrayed in Fig. 168. Each *P* wave is followed by a ventricular complex; each contraction of the auricle stimulated the ventricle to respond. Measurements show, however, that there is a delay in auriculoventricular condition, the *P-R* time averaging about 0.25 of a second. But the interference causing this delay was not great enough to prevent any of the auricular impulses from reaching the ventricle.

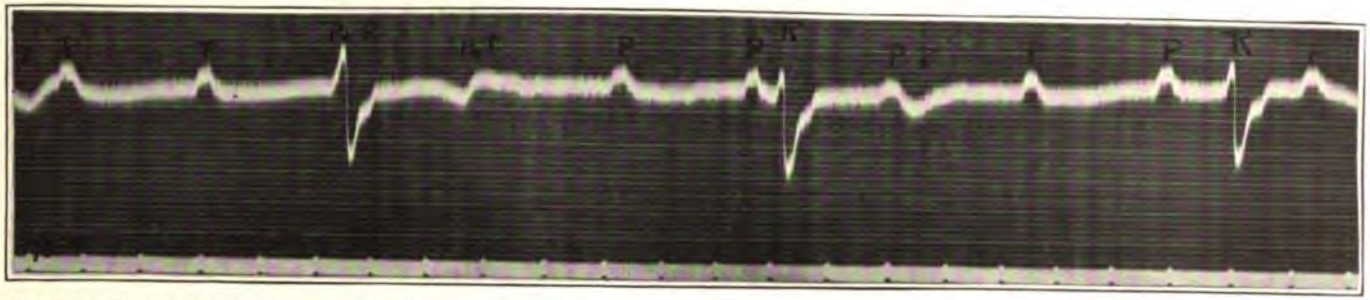


Fig. 167.—Electrocardiogram of complete heart block. *P* waves occur rhythmically. Ventricular complexes also occur rhythmically but with no relationship to auricular waves.

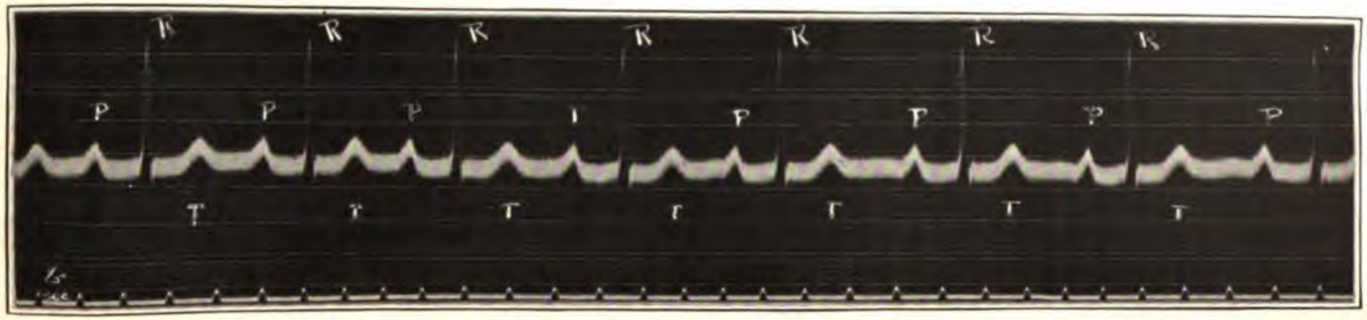


Fig. 168.—Electrocardiogram of delayed conduction. All *P* waves are followed by ventricular complexes, but only after a prolonged time interval. The conduction time averages about 0.25 of a second.

Whether the interference to a-v conduction results, then, in delay, in partial or complete block, depends upon the degree of interference to the passage of impulses. This varies under many influences, and this variation may upon occasion result in the occurrence of more than one of these conditions in the same subject within short periods of time. For the sake of clearness, however, each will be considered singly in discussing the signs that indicate its presence.

Partial Heart Block.—In the case of a ventricle that is contracting in response to impulses that are being sent at regular intervals from the auricle, the failure of one of these impulses to reach the ventricle will result in a ventricular pause and an interruption to the rhythm. At that moment no apical thrust against the chest wall occurs, no heart sounds are heard and no pulse is felt. There is a ventricular silence, a "dropped beat." The pause continues until the next rhythmic auricular impulse reaches the ventricle and causes it to contract. The character of the arrhythmia in a given case will depend upon the frequency and regularity of these dropped beats, i.e., upon the frequency and regularity with which auricular impulses are blocked.

If every second impulse from the auricle fails to reach the ventricle, the rate of the latter will be halved; but, since it is beating in response to alternate rhythmic impulses, no arrhythmia will be produced. Each beat of the ventricle produces a peripheral pulse. Since there is no arrhythmia, this condition, if it persists, rarely admits of certain diagnosis without instrumental means. It is rarely persistent, however, and usually shows frequent transitions either to normal rhythm or to other forms of partial block. An abrupt doubling or exact halving of apex rate is quite suggestive. The probability increases if transitions to other forms of partial block occur.

If every third auricular impulse is blocked, the resulting ventricular silences produce a coupled rhythm, a *bigeminy* both of apex and of pulse. Blocking of every fourth auricular stimulus produces a trigeminy of apex and pulse. In like manner different numbers of ventricular beats may be set off by pauses, and the number so marked off may vary within short periods of time.

As in the case of sinus arrhythmia and premature contractions, considerable increase of pulse rate, as by exercise, usually causes the disappearance of partial blocking and produces regularity of the ventricle.

In cases of mitral stenosis, partial heart block may produce striking changes in the murmurs heard at the apex. The blocked auricular systole may produce a murmur similar to the presystolic accentuation heard with other beats. The time relationship of this murmur to the heart sounds will depend upon the relationship of the blocked auricular systole to the next succeeding ventricular systole.

Differential Diagnosis

It is to be noted that premature contractions may occur with such frequency or regularity as to produce a pulse not unlike that of partial block. Thus a bigeminal pulse may be produced by the regular occurrence, after every second normal beat, of a premature beat so weak that no radial response is produced by it. In the case of such premature beats, however, evidences of ventricular activity are present at the apex during the pulse pause, while in the case of partial block the ventricle is silent during the intermission. A bigeminal pulse may be produced by premature contractions in another way. After each normal beat a premature beat of sufficient strength to reach the wrist may occur. The pauses following these premature beats will set them off in pairs. In such a case, however, the second pulse is weaker, and the heart sounds produced by the premature beat also are weaker.

The differentiation of occasional ventricular intermissions from sinus arrhythmia is to be accomplished by noting the effect of respiration upon the latter.

Complete Heart Block.—Since the ventricle, deprived of auricular stimuli, establishes its own rhythm in this condition, its beats occur regularly (as a rule) and slowly. The rate is usually under 40, though not infrequently higher rates are observed. Both heart sounds are heard with each systole, and each contraction produces a radial pulse.

Under favorable conditions the examiner may be able to hear the sounds produced by the contractions of the auricle. These, when audible, are heard occurring rhythmically and with no constant relationship to the regular heart sounds produced by the ventricle. Their rate, derived, as it is, from the pacemaker, is more rapid than that of the ventricle, and is about that of the normal heart.

Even in cases in which the rhythmic auricular sounds cannot be demonstrated, their occurrence may modify the sounds produced by the ventricle. When a contraction of the auricle occurs simultaneously with the first or second heart sound, it duce a noticeable increase in intensity of the sound w place at the same time with it. Its occurrence just first, or immediately following the second sound may "reduplication" of the sound near which it occurs.

The rhythmic auricular systoles produce small jugu tions which may be visible in the neck during ventricula When one of these waves coincides with that produce tricular systole, i. e., when auricular and ventricular sy together, a visible accentuation of venous pulse may

Delayed Conduction.—Since each heart beat arises and stimulates the ventricle to a normal response, the be latter occur rhythmically, and at a rate determined by maker. It is impossible, therefore, to establish a dia delayed conduction without instrumental means of meas auriculoventricular interval.

The condition is most common, however, in hearts w are the seat of mitral disease. In cases which show t acteristic diastolic murmur and thrill with presystolic a tion, these signs may be modified by a delay in a-v cond a way which strongly suggests, if it does not prove, the conduction. The auricular systole, normally occurring ju ventricular systole (first heart sound) is responsible for systolic accentuation of murmur and thrill in mitral If, then, the time relationship of auricular systole to ve systole is altered, (by a lengthening of the a-v inter systole of the auricle will occur not just before ventric tole (presystolic), but earlier, i. e., in late, middle diastole, depending upon the length of the a-v interv change in position of the auricular systole by the length interval, will cause a corresponding change in the po diastole which the accentuated murmur assumes. The then, may be best heard and the thrill best felt, not ju ventricular systole but earlier in diastole. Such a relati diastolic murmur to ventricular systole in cases of mitra is always quite suggestive of delayed conduction.

The same altered relationship of auricular systole to s the ventricle, separating these contractions by an interv than normal, may modify the heart sounds in cases with lesion. The occurrence of auricular systole earlier in than normal may result in its being heard as a "redup of the first heart sound. Its occurrence still earlier in ve diastole may cause a "reduplication" of the second heart sound.

Symptoms of Heart Block.—The milder degrees of heart block usually cause no symptoms which are attributable directly to the blocking of auricular impulses.

In the severer grades, periods of excessive ventricular slowing are likely to occur; and such slowing of the ventricle accounts for the grave symptoms which frequently accompany this condition. Such attacks often exhibit an irregularity as well as a slowing of the ventricle. Dizziness, faintness, syncope may occur, depending upon the length of time during which the brain is unsupplied with blood. Convulsive seizures, and not uncommonly death, may result.

AURICULAR FLUTTER

The pacemaker, held in check by the vagi, under normal conditions initiates contractions of the auricular musculature which vary in rate within rather wide limits, depending upon the degree of vagus tone which changes with exercise, excitement and many other influences. Even with a constant rate, small variations in the lengths of the interauricular intervals are present.

Under certain abnormal conditions, however, auricular movements take place at a rate far above normal limits and with great regularity. These contractions are not under the influences that are usually exercised at the pacemaker. The rate is constant and the degree of rhythmicity is extremely high. To this condition of rapid and regular contractions of auricular musculature the term *auricular flutter* has been applied.

There is much evidence to indicate that these movements of the auricle arise in a circus movement that has become established in a natural ring of auricular musculature. Whatever the cause, the condition is characterized by rhythmic auricular movements of high rate, usually about 300 per minute; it tends to persist for long periods of time, and the rate is remarkably constant.

There is almost always a certain amount of auriculoventricular blocking, the most common form consisting of 2:1 block. The rate of the ventricle, therefore, is lower than that of the auricle, and may be one-half, one-fourth or some other fraction of the auricular rate.

Fig. 169 is an electrocardiogram of auricular flutter with 4:1 block. The broad, downwardly deflected waves represent auricular contractions; the sharp upwardly directed waves, lettered *R*, represent activity of the ventricular musculature. One auricular wave coincides with each *R* wave and is partly obscured by it. Following each *R* wave is a deflection deeper than the others. This is produced by a blending of the *T* wave of ventricular activity with the auricular wave that occurs synchronously with it. It will be noted that the ventricular complexes occur regularly with every fourth auricular wave. The calculated ventricular rate is 61 per minute, the rate of the auricle 244. An examiner, upon observing the patient at the time this record was being made, would have noted, then, an apex and pulse rate of 61, both occurring regularly. There probably would have been observed no signs of abnormal cardiac mechanism, and the diagnosis would have depended upon the electrocardiogram or upon other instrumental means.

Usually, however, there are clinical indications which suggest the presence of flutter and in many instances render the diagnosis fairly certain.

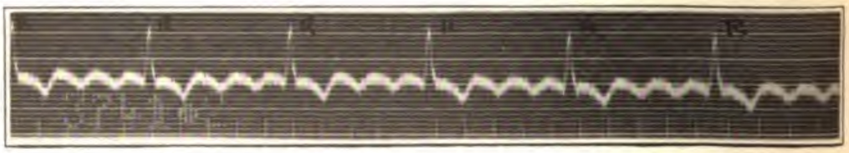


Fig. 169.—Electrocardiogram of auricular flutter. Four auricular waves occur to one ventricular complex. The *R* and *T* waves cause distortion of auricular waves that are synchronous with them.

Since the auricular rate is rapid and 2:1 block is most common, the observed rate of pulse and apex is commonly high, 120 to 150 or more. Since, also, the condition tends to persist, the observed ventricular rate usually *remains at the same constant high level* for weeks or months. And since the pacemaker exercises no control over the rate, the latter is uninfluenced by exercise, posture, etc. The condition is most common in individuals of advancing years who also show evidences of arterial sclerosis. The pulse is usually regular, but irregularity is not infrequent since the blocking of auricular impulses does not always take place with regularity. Such arrhythmias are difficult to diagnose without graphic curves. The ventricular beats, taking place as they do in response to rhythmic impulses from the auricle, occur at intervals which may be anticipated by taking account of the rhythm. But this is difficult to accomplish with accuracy without instrumental means. Exercise, however, of small amount may cause the disappearance of this arrhythmia, and produce the un-varying rate which is characteristic of auricular flutter. There may be a history of attacks of "palpitation" caused by such short periods of flutter.

Occasionally each auricular contraction may produce a beat of the ventricle for short periods; there being, for a time, no blocking of the auricular impulses. The subject of such attacks usually loses consciousness until a resumption of partial block slows the ventricular rate.

AURICULAR FIBRILLATION

The normal systole of the auricle is an orderly and coordinated affair. The contraction spreads from pacemaker to a-v node, involving successive portions of auricular musculature in sequence; and by producing, for an interval, synchronous shortening of all muscle parts, expels blood from the chamber.

Under certain pathologic conditions this orderly sequence of events does not take place. The auricular muscle quivers and twitches in various places in extreme disorder. No concerted action, no synchronous shortening of all muscle parts, occurs. There is no systole of the whole chamber, and blood is not expelled. The auricular activity appears to consist of rapid fibrillary twitchings in all parts of the musculature. This disordered auricular action is called *auricular fibrillation*. It has a close association clinically with auricular flutter, and the explanation of this uncoordinated activity which at present appears most reasonable is that it, too, is produced by circus movement in auricular muscle. Instead of a ring of auricular muscle undergoing simple contractions, it is supposed that depressed conduction and, in addition, local variations in conduction have so broken up the waves from the ring movements that many complex waves find their way through the auricular musculature. Whatever the cause, the result is disordered action of the auricle which affects ventricular activity in a characteristic way.

The ventricle, no longer receiving rhythmic stimuli of constant type from the auricle, now receives stimuli which vary in strength as well as in periodicity. Instead of being allowed a period of rest, for recuperation and preparation for the next beat, the ventricle must respond as best it may to the various impulses that crowd down upon it. Unless there is also some interference to a-v conduction, these impulses arrive at rapid rate. Some find the ventricle little prepared to contract. Others ar-

[illegible]

[illegible]

[illegible] the supplying of [illegible] the atria to contract, there[illegible] little importance [illegible] its normal rhythmic [illegible] is of importance chiefly because [illegible]. It is, indeed, an indication of [illegible] raises the question [illegible] be confined to that chamber. But its effects [illegible] are responsible for the symptoms which it produces. The ventricle, except in cases with impaired [illegible], is constantly being goaded by rapidly arriving impulses. It is deprived of its regular periods of rest. This overwork taxes its reserve, and, in cases in which the latter has already been drawn upon, may be enough to induce decompensation or to add further symptoms if decompensation is already present. The patient may be conscious of the irregularity in the force and rhythm of the ventricular beats, and these be may refer to as "palpitation." Occasionally there are brief attacks

of fibrillation, and these, of course, produce only brief sympton
but as a rule the condition is of long standing, once it has b
come established.

PAROXYSMAL TACHYCARDIA

Diminution of vagus tone at the sinus area with exercise, e
citement, fever and other conditions allows stimuli to be fre
at the pacemaker which cause contractions of the heart in norm
fashion at a rate which may reach 160 or more per minu
Under such circumstances the rate is lowered by an increase
vagus tone at the pacemaker, and may show considerable vari
tions within short periods of time.

A tachycardia which is produced by lowered tone at the pac
maker is called a *simple tachycardia*. Although the rate may
high, it is subject to the same influences that are normally e
hibited in controlling heart rate. The acceleration may ta
place quickly but it is not entirely abrupt. The rapid rate m
show further variations while maintained at a high level. T
return of the rate to its usual level is rather gradual.

Another type of tachycardia is not infrequently met wit
Some ectopic focus suddenly begins to elaborate and free i
pulses to contraction at a rapid rate. Not being under the co
trol that is normally exercised by the vagus at the pacemak
the rate at which this ectopic focus sends forth stimuli shows
variation with vagus influence. It is constant. The new foc
suddenly assumes the rôle of pacemaker and as abruptly yiel
again its new rôle to the normal area. This ectopic focus in mo
cases is in the auricle, though occasionally a focus in the ve
tricle or in the a-v connecting tissue becomes such a center
abnormal stimulus production.

There is this relationship between premature contractions a
paroxysms of tachycardia: both originate in an ectopic foc
which elaborates stimuli more rapidly than it does under norm
conditions. Cases which are subject to paroxysms of tachycard
frequently exhibit, during the interval between attacks, pr
mature contractions whose electrocardiographic form indicat
that they arise in the same focus whence the tachycardia ori
nates.

Fig. 171 is an electrocardiogram from a case of paroxysm
tachycardia with origin in the auricle. With each cycle t
auricular wave P is superimposed on the T wave of the precedi

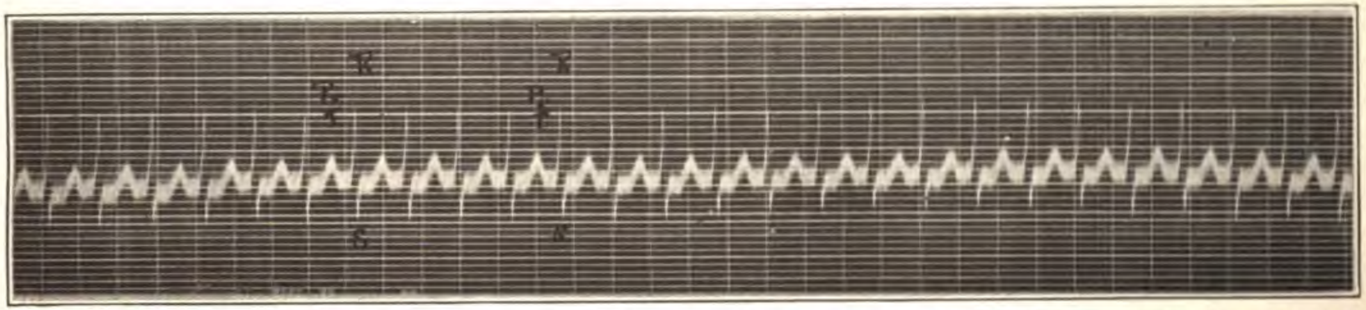


Fig. 171.—Electrocardiogram of paroxysmal tachycardia with origin in the auricle. Ventricular complexes are of supraventricular form. *P* and *T* waves are superimposed. Rate 222.

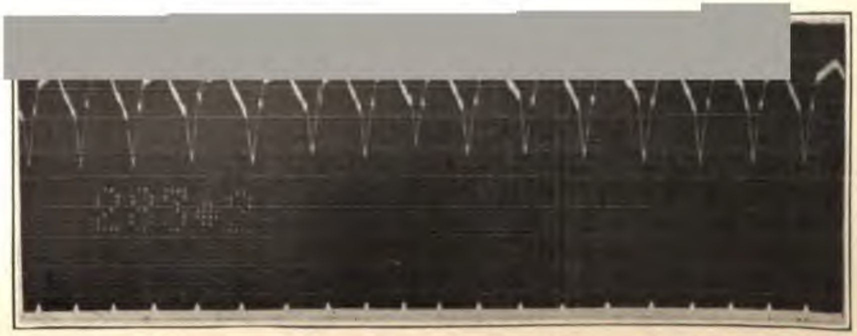


Fig. 172.—Electrocardiogram of paroxysmal tachycardia with origin in the ventricle. The ventricular complexes are of a type different from those which occur in response to an auricular stimulus. They occur rhythmically. Traces of auricular waves are seen at points lettered *P*.

ventricular complex and both are seen as one broad upward deflection. The ventricular complexes are of type similar to others which are produced by stimuli that come from the auricle; i. e., they are of supraventricular form.

Fig. 172 is also an electrocardiogram from a case of paroxysmal tachycardia, but in this case the abnormal focus of stimulus production was in the ventricle. The ventricular complexes, therefore, are of a type different from those in Fig. 171. The auricular waves are not well shown, being superimposed on the ventricular waves. Traces of them are clearly seen, however, as modifications of the ventricular waves. They are lettered *P*. The ventricular rate is about 220, the rate of the auricle about 115.

The Signs and Symptoms of Paroxysmal Tachycardia.—The attacks or "paroxysms" of tachycardia begin and end abruptly. This is perhaps the most characteristic feature. The termination may be marked by the occurrence of a few irregularly spaced beats before the normal rhythm is assumed. Except in the case of paroxysms of quite brief duration, the patient is conscious of the onset and ending of the attack, and while it lasts he feels the pounding of the heart. The rate during an attack is remarkably constant and is not influenced by posture, exercise, etc. It is usually found to be between 140 and 190, though rates above and below those figures are observed.

The paroxysm may be brought on by exertion, emotion or other influence or it may occur spontaneously. The duration is variable. It may last for only a few beats, or it may persist for days. Most commonly it is a matter of several hours.

Paroxysms of brief duration usually cause few symptoms other than a consciousness of rapid and forceful heart action and usually a sense of weakness. With longer attacks, however, more serious consequences usually follow. The feeling of weakness and faintness increases. Gastric symptoms often ensue. It can sometimes be demonstrated that the heart has dilated to some extent. The blood pressure falls. The heart sounds, which have assumed a tic tac quality, may become fainter with the longer duration of the attack. Congestion and edema of the lungs with frothy, bloody sputum, and other signs of failing heart may ensue. In most instances the attack has subsided before these more serious symptoms make their appearance. The patient then experiences a sense of relief and rapidly recovers from the symptoms produced by the paroxysm.

Many subjects of paroxysmal tachycardia learn certain measures which cut short the attack with more or less constancy. One may cause a cessation by taking and holding a deep breath, and especially by raising the diaphragm with the glottis closed after a full inspiration. Another makes strong pressure in the epigastrium, another bends forward and downward; thus producing the same effect. Other measures include the taking of gastric irritants and the induction of emesis. Other forms of vagus stimulation, such as direct pressure of the carotid, may stop an attack. Such measures, however, do not cause slowing, as they may do in simple tachycardia. They either stop the paroxysm or do not affect the rate.

There is usually a sense of oppression in the chest, and not unusually anginal pains occur, especially in the longer paroxysms. Death is a relatively infrequent ending of attacks, but is not altogether uncommon with those of long duration.

PULSUS ALTERNANS

The contractions of the ventricle under normal conditions are of equal force. Certain hearts, however, which are accomplishing their work with difficulty, exhibit an alternate variation in the strength of individual beats while maintaining a regular rhythm. This occurs particularly in cases of sclerosis and high tension which give other signs of failing myocardium. The ventricle expels alternately variable quantities of blood. The exact cause is unknown.

The difference in force may be great enough to be appreciated upon feeling the pulse. By careful palpation the alternate variation can often be detected in this way even though the difference is slight. The diagnosis may be exactly made by applying the sleeve of a sphygmomanometer to the arm and noting that there is one level of pressure at which the weaker beats disappear and a higher one for the alternate beats. By thus applying the correct pressure the arterial rate may be exactly halved.

Occasionally the difference in force is so great that the pulse rate is halved, the smaller beats not being perceived. This, ho ever, is quite rare. The heart sounds exhibit no difference quality. A pulse tracing shows the alternation in the height the arterial waves. An electrocardiogram cannot be depended on to show any indication of the condition. It often does show an alternate variation in the height of the *R* waves, but more commonly, perhaps, it does not. On the other hand the electrocardiograph sometimes shows alternation in a heart which gives no clinical evidence of it.

Fig. 173 is an electrocardiogram in which the alternation is depicted. There is a slight, though definite, alternation in the height of the *R* waves. To insure clearer reproduction in the text, the peaks of the *R* waves in this figure have been indicated by short horizontal lines.

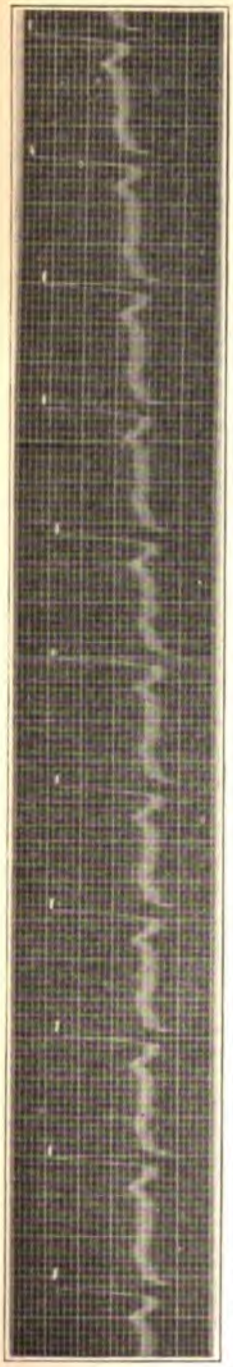


Fig. 173.—Electrocardiogram of pulsus alternans. Short horizontal lines are drawn at the level of the peak of each *R* wave. Note the alternate variation in the height of the *R* waves.

DIAGNOSTIC THESES

In attempting to diagnose abnormalities of cardiac mechanism without instrumental means certain generalizations should be kept in mind. It is of value to examine both heart and pulse: of these more may be learned from the heart. When the heart action is irregular it is important to determine whether there is a *predominant rhythm;* i. e., a rhythm which, irrespective of interruptions, dominates the action as a whole. If no such predominant rhythm can be demonstrated, the arrhythmia is probably due to auricular fibrillation. This probability increases if the rate is high.

It should be determined whether each ventricular beat produces a radial pulse. Neither heart block nor sinus arrhythmia produces a *pulse deficit.* Auricular fibrillation usually does so; many premature beats fail to reach the wrist; and occasionally an alternating pulse may give a pulse rate just half that of the ventricle.

In doubtful cases with slow rate an attempt should be made to accelerate the action of the ventricle. If the arrhythmia disappears with higher rate it is not due to auricular fibrillation; if it becomes more pronounced the probability of fibrillation is increased.

Occasional interruptions to an established rhythm are probably due to premature contractions. A regularly recurring interruption is probably due to premature contractions or to partial heart block, more likely to the former.

A bigeminal pulse may be produced by premature contractions which recur regularly after every second beat and fail to reach the wrist, or by the occurrence of a premature contraction which reaches the wrist after every normal beat. It may be produced by a 3:2 heart block.

An exact halving of an observed pulse rate may be produced as follows: (a) by the occurrence after every normal beat of a ventricular premature contraction too weak to reach the wrist; (b) by the onset of a 2:1 heart block; (c) by the change from a 2:1 auricular flutter to a 4:1 type; (d) by the onset of pulsus alternans of extreme degree.

A regular ventricular action which remains of constant rate from day to day, and which is uninfluenced by exercise and posture is probably due to flutter or to paroxysmal tachycardia. If the rate is less than 100 it is almost surely 4:1 flutter. Paroxysms of tachycardia are usually of brief duration; flutter usually persists.

SECTION V

DISEASES OF THE CIRCULATORY ORGANS

CHAPTER XVIII

DISEASES OF THE PERICARDIUM

PERICARDITIS

Inflammation of the pericardium occurs primarily and as a secondary disease. Primary pericarditis has followed trauma to the pericardium from external violence. Certain cases of primary so-called idiopathic pericarditis develop in children without assignable cause.

Secondary pericarditis occurs as a sequence of acute rheumatic fever, acute tonsillitis and other septic states, as also in gout, tuberculosis, and during the course of general arteriosclerosis.

Pericarditis occurs in three principal forms; namely, *acute fibrinous pericarditis; serofibrinous pericarditis,* or pericarditis with effusion; and *chronic adhesive pericarditis.*

ACUTE FIBRINOUS PERICARDITIS (PERICARDITIS SICCA)

Clinical Pathology.—In this form of pericarditis, which is also termed pericarditis sicca, the surface of the visceral pericardium, and later during the evolution of the disease that of the parietal pericardium as well loses its normal smooth, glistening appearance and becomes roughened. It is the seat of a fibrinous exudate, which may be circumscribed to a small portion of the membrane, or which may be universal, involving the entire pericardial surface. There is usually slight effusion into the pericardial sac, but in cases of tuberculous origin it is distinctly a dry inflammation. The roughened, exudate-clothed pericardium does not glide noiselessly as is the case during health, but in lieu of this its measurements are accompanied by an audible friction rub.

The gross appearance of the pericardial surface varies in different stages of the disease. In the incipiency of the disease the surfaces present the appearance of two slices of bread and butter which have been apposed and pulled asunder, the "bread and butter" stage of the disease. In other instances the exudate is rolled into irregular folds upon the pericardial surface, constituting the "cor villosum."

The subjacent myocardium is involved to a variable extent in the inflammatory process, presenting infiltration with leucocytes, and endocarditis is occasionally a coincident disease.

Physical Signs.—*Inspection.*—In acute fibrinous pericarditis inspection usually reveals few signs, though it is not uncommon for the respirations to be accelerated, indeed occasionally to the point of orthopnea. The cardiac impulse is accentuated in force, and frequently its area is extended. The facial expression is apt to be anxious.

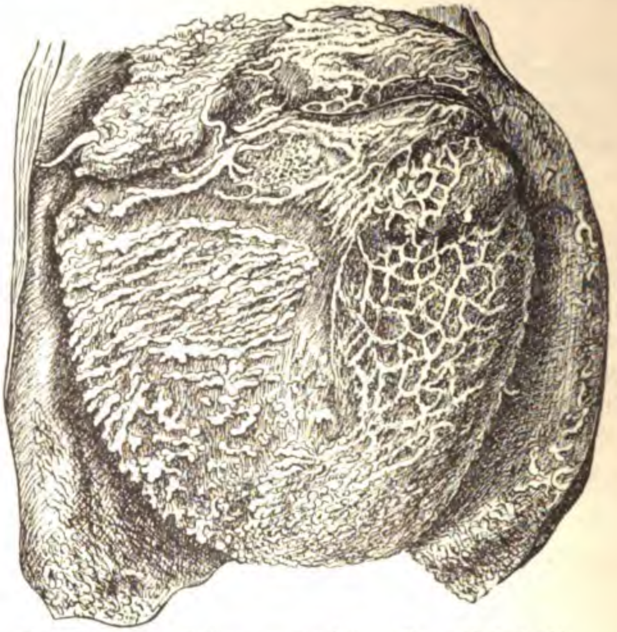


Fig. 174.—Acute fibrinous pericarditis. (From McFarland.)

Palpation in the fully established cases frequently reveals the presence of pericardial friction fremitus over the precordia, which is increased in intensity when the patient bends the trunk forward. The pulse is accelerated, but is usually of normal volume and tension.

Percussion reveals no deviation from the normal outline of the heart, as the organ is not enlarged in uncomplicated acute fibrinous pericarditis.

Auscultation elicits the pericardial friction sound over the precordia. This sound is variable; it is increased by moderate pressure and is abolished by strong pressure with the bell of the stethoscope; it also varies in intensity upon change of posture; and it varies in intensity from day to day without apparent cause. The sound corresponds roughly with the sounds of the heart, but not with the same accuracy as do endocardial murmurs, as the phases of the friction sound last longer than do the heart sounds. The friction sound usually presents two phases, systolic and diastolic, occasionally only a single phase, and in yet other instances three phases, simulating canter-rhythm or gallop-rhythm. When the sound presents the typical systolic and diastolic phases, these phases exhibit the same intensity, but are usually of unequal length. The pericardial friction sound is confined strictly to the precordia and is not transmitted thence, as are endocardial murmurs. If the associated effusion becomes great, the friction sound disappears, but not infrequently it remains audible over the base of the heart even at the height of the effusion. The sound is usually heard with the greatest ease along the left border of the sternum in the fourth and fifth interspaces, or near the aortic valve area. In certain instances, however, the sound is most clearly audible over the cardiac apex. The pericardial friction sound seems very superficial, as if it were just beneath the ear.

Diagnosis.—The diagnosis of acute fibrinous pericarditis is readily made when the disease is suspected and the friction sound, the pathognomonic sign of the disease, is detected. But, arising as it so frequently does during the course of an acute infectious disease, it is often masked by the other symptoms of the disease and is not suspected or sought for and hence is frequently overlooked.

Differential Diagnosis.—A mistake in diagnosis may arise in failing to differentiate acute fibrinous pericarditis from organic disease of the aortic valve, or from pleuropericardial friction in pleurisy arising during pneumonia or pulmonary tuberculosis. Organic disease of the aortic valve produces a double murmur, systolic and diastolic in time, with a palpable thrill, which may be easily mistaken for the two phases of the pericardial friction sound. But this double murmur corresponds very closely indeed with the events of the cardiac cycle; the murmurs are transmitted from the precordia in different and definite lines; and the disease produces alterations in the character of the pulse, as for instance the water-hammer pulse in Corrigan's disease, and the small wiry pulse of aortic stenosis. The close circumscription of the maximum intensity of the murmur to the aortic orifice is of aid i differentiation; and, moreover, aortic disease causes hypertroph of the heart with alteration in the cardiac outline. The pericardial friction sound is more superficial and variable than is th aortic murmur, and is influenced by pressure with the bell of th stethoscope, which does not influence the murmur of organic disease of the aortic valve.

The pleuropericardial friction sound resembles in some respects the pericardial friction sound, but it disappears during suspension of respiration following full inspiration.

Acute fibrinous pericarditis is accompanied by pain over the precordia or around the xyphoid appendix, and by moderate fever.

SEROFIBRINOUS PERICARDITIS (PERICARDITIS WITH EFFUSION; PERICARDITIS EXUDATIVA)

Clinical Pathology.—Serofibrinous pericarditis, pericarditis with effusion, or pericarditis exudativa has its inception as a dry plastic pericardial inflammation, during which stage of the disease the pericardial surface is clothed with fibrinous exudate. In the further evolution of the disease there is effusion of serofibrinous fluid, which fills the dependent portions of the pericardial sac. The fluid is frequently turbid, containing flocculi of fibrin. The quantity of the fluid varies from a few ounces to two liters or even more in the extreme case.

Unless aspirated, the natural tendency is toward absorption of the fluid, occasionally with no permanent damage to the pericardium, but in other instances with the formation of adhesions between the visceral and parietal layers of the membrane; and [illegible] instances with localized areas of pericardial thickening, the so-called "milk spots," or "soldier spots" of this [illegible]

[illegible] is inflamed to a variable degree, and [illegible] a concomitant condition.

[illegible]—Serofibrinous pericarditis is at- [illegible] precordial bulging, particularly when [illegible] childhood, when the chest wall is very [illegible] subject with thin thoracic parietes. [illegible] male does not invariably yield to [illegible] tension. However, in the presence of extensive effusion into the pericardium there is visible epigastric bulging in the vast majority of cases, constituting Auenbrugger's sign of the disease. In a fully established effusion the cardiac impulse is not infrequently invisible; or, when it is visible, the impulse is commonly displaced upward, presenting a wavy pulsation in the third or fourth intercostal space. When the apex beat is invisible with the patient in the recumbent or sitting posture, it may in certain instances become again visible when the patient bends the trunk forward.

The respirations are moderately accelerated, and in extensive pericardial effusion they are frequently attended by dyspnea which occasionally amounts to orthopnea. The left half of the thorax is apt to expand in a deficient fashion, as a result of compression of the left lung by a large effusion. Rarely, indeed, does the examiner encounter tortuosity of the superficial veins of the thorax as a result of increased intrathoracic tension.

Palpation.—Palpation confirms the displacement of the cardiac impulse. Early in the disease a friction fremitus is palpable, which disappears as the effusion develops, though it sometimes persists at the base, to reappear with absorption of the effusion. As to intensity, the cardiac impulse is feeble, and gradually decreases in intensity as the effusion develops, to eventually disappear altogether. Ewart has noted that the first rib is freely palpable at its chondrosternal articulation in pericarditis with effusion, the "first rib sign." Fluctuation can be obtained only in the presence of very large effusions and is not to be expected in other cases. Frequently there is moderate tenderness upon palpation of the epigastrium.

The pulse in serofibrinous pericarditis is usually weak and of small volume, often irregular, and of the pulsus paradoxus type, becoming progressively more feeble during full inspiration.

Percussion.—The area of cardiac dullness is extended, and this increases progressively as the effusion develops. The area of dullness is roughly pear-shaped, with the base directed downward toward the diaphragm. In the fifth intercostal space the dullness extends one or two inches to the right of the sternum, obscuring the normal pulmonary resonance in Ebstein's cardiohepatic angle, constituting Rotch's sign of the disease. Upon the left side the dullness of the heart is apt to extend outward beyond the apex or even to encroach upon and obscure the normal gastric tympany in Traube's semilunar space. The diaphragm and the left lobe of the liver are depressed by the weight of the effusion. Gerhardt pointed out that when the patient is in the upright posture the area of dullness is broader than when he is in the recumbent posture. A coexisting hypertrophic emphysema may serve to obscure the dullness of a fairly large pericardial effusion by the interposition of the voluminous anterior pulmonary borders between the pericardium and the anterior wall of the thorax.

Percussion of the left side of the thorax posteriorly, near the angle of the scapula, with the patient in the upright posture, in the presence of extensive pericardial effusion not infrequently yields dullness, which disappears when the patient inclines the trunk forward, Bamberger's sign of the disease.

Auscultation.—During the incipient stage of the disease, prior to the development of the effusion, a pericardial friction sound is audible over the precordia. When the effusion develops this friction rub disappears gradually, although frequently persisting at the base of the heart. If inaudible when the patient is in the recumbent posture, it occasionally appears when he is placed in the sitting posture. The cardiac sounds become gradually weakened and muffled as the effusion develops. The pulmonic second sound is apt to be accentuated. The compression of the left lung in extensive pericardial effusion is apt to result in bronchovesicular or bronchial breath sounds in the axillary region.

Diagnosis.—The pear-shaped area of increased cardiac dullness, the initial friction sound, which disappears with the development of the effusion, the muffled and distant character of the cardiac sounds, with pain over the precordia, which is aggravated by pressure over the lower end of the sternum, dyspnea, occasionally cyanosis, the paradoxical pulse, and fever, point strongly to effusion into the pericardium.

Differential Diagnosis.—Dilatation of the heart produces increase in the area of the heart, but in this disease there are sufficient differential signs to distinguish the two conditions. In cardiac dilatation the cardiac impulse instead of being obscured is distinctly visible and wavy; the shock of the cardiac valves is distinctly palpable in dilatation; the area of cardiac dullness in dilatation is not pear-shaped, neither does it, except in the case of mitral stenosis, reach so high along the left sternal margin or so low in the fifth and sixth interspaces without visible or palpable impulse; rarely indeed is the enlargement of the heart in dilatation so large as to compress the left lung with the production of dullness and bronchovesicular or bronchial breath sounds over the left lung; in dilatation the cardiac sounds are distinctly audible, frequently with a valvular quality engrafted upon them; and the fluoroscopic findings are entirely different in the two diseases.

Serofibrinous pleurisy occasionally must be differentiated from pericarditis with effusion when the effusion reaches an extreme grade. In left-sided pleural effusion, unless the effusion assumes the encysted form, it is likely during a casual examination to be mistaken for a large pericardial effusion; and similarly a large pericardial effusion may be difficult to differentiate from effusion into the left pleural sac. But in pleurisy with effusion the cardiac impulse is displaced; the flatness extends around the side of the thorax; the compressed lung yields Skodaic resonance in the infraclavicular and mammary regions; Traube's similunar space of gastric tympany is obliterated; and the spleen is apt to be displaced downward. In serofibrinous pericarditis, on the contrary, the effusion is apt to extend well to the right of the sternum (Rotch's sign), which is of aid in the differentiation of the two diseases.

CHRONIC ADHESIVE PERICARDITIS

Clinical Pathology.—In chronic adhesive pericarditis adhesions are formed as the consequence of a previous acute pericarditis of the fibrinous or serofibrinous form. In many instances the adhesions are sparsely distributed or are localized to a limited area of the visceral and parietal membranes, while in other cases they are more or less universally distributed over these membranes. Moreover, the adhesions may exist between the external surface of the pericardium and the adjacent pleura, constituting pleuropericarditis or mediastinopericarditis.

The internal form of chronic adhesive pericarditis, in which the adhesions exist between the visceral and parietal layers of the membrane, often does not lead to embarrassment of the cardiac action, and frequently gives rise to no symptoms and few physical signs. But when the adhesions are abundant there is a variable degree of cardiac embarrassment, with consequent hypertrophy of the heart.

In external chronic adhesive pericarditis adhesions bind the outer surface of the pericardial sac to the costal pleura anteriorly or to the diaphragm, or to the esophagus or spinal column, or to the great vessels arising from the base of the heart. In this form of t ase the contraction of the ventricles not infrequently lic retraction of the thoracic wall; or the constricting bands may result in dysphagia, stridulous respiration, or vascular murmurs.

The coincident changes in the myocardium in chronic adhesive pericarditis vary with the degree of interference with the heart's action. Frequently it is entirely unaltered in the presence of simple adhesions within the pericardial sac; while it presents hypertrophy and occasionally thinning and dilatation of the walls of the cardiac chambers in the presence of extensive adhesions.

Physical Signs.—*Inspection.*—In cases which are associated with cardiac hypertrophy there is apt to be moderate precordial bulging, with displacement of the cardiac impulse from its normal site in the fifth left intercostal space. There is frequently systolic retraction of the chest wall anteriorly near the apical area. In cases with diaphragmatic adhesions there is often a systolic retraction of the tenth and eleventh interspaces posteriorly below the left scapula (Broadbent's sign).

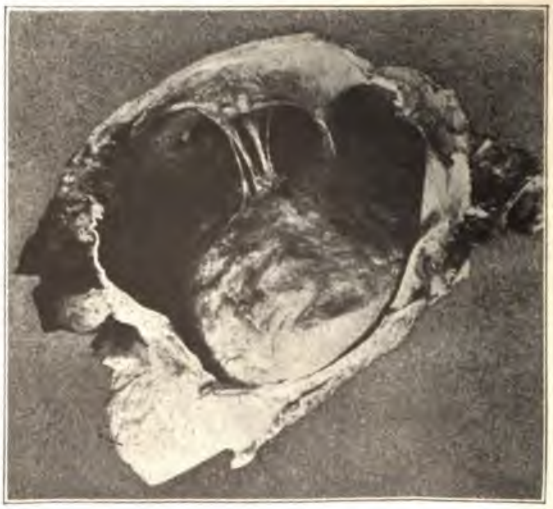


Fig. 175.—Pericardial adhesions. (From Delafield and Prudden.)

Friedreich's sign, diastolic collapse of the jugular veins is noted frequently; and Kussmaul's sign, inspiratory overfullness of the jugulars is occasionally in evidence. The diaphragmatic shadow is frequently restricted or abolished by partial immobilization of the diaphragm by adhesions. The apex-beat, in addition to the displacements to which it is so frequently subject in this disease, does not exhibit its normal range of lateral mobility upon placing the patient in the lateral decubitus.

Palpation.—In chronic adhesive pericarditis there is not infrequently demonstrable upon palpation of the precordia a distinct diastolic shock, which is due to the sudden rebound of the heart walls during diastole, which have been drawn into apposition during systole increasing the tension which is exerted by the adhesions. Palpation is useful in defining the displacement of the cardiac impulse. Adhesions between the pericardium and diaphragm frequently inhibit the normal epigastric excursion during inspiration. The pulse is of the pulsus paradoxus type in many cases, trailing off during full inspiration.

Percussion.—As a rule, percussion reveals a considerable increase in the transverse diameter of the area of cardiac dullness; but this is not constant, as in certain cases the heart is neither hypertrophied nor dilated. Occasionally the superior and left borders of the area of cardiac dullness are not diminished at the completion of full inspiration, because adhesions between the pleura and the pericardium prevent the intervention of the anterior border of the left lung between the heart and the anterior chest wall during inspiration. Not infrequently the gastric tympany of Traube's semilunar space is diminished.

Auscultation.—The character of the heart sounds varies with the state of the myocardium. In the presence of coincident cardiac hypertrophy they are accentuated; whereas, if cardiac dilatation has supervened, they are muffled and more or less valvular: Murmurs of coexisting valvular disease are occasionally audible, which are not dependent upon the disease for their production. Occasionally a pericardial friction sound is audible along the left sternal border. Reduplication of the second sound of the heart is occasionally audible, and sometimes there is a soft blowing systolic murmur at the mitral area.

Diagnosis.—With a history of previous pericarditis, the finding of signs of pericardial adhesions, such as fixation of the cardiac impulse, inspiratory overfullness of the jugular veins and diastolic collapse of these vessels, with systolic retraction of the thoracic wall anteriorly, or Broadbent's sign posteriorly, is suggestive of chronic adhesive pericarditis. While it is true that a systolic retraction of the chest wall in the region of the cardiac apex may be due to atmospheric pressure in the presence of cardiac hypertrophy, when the anterior borders of the lungs are tardy in closing the interval between the heart and the anterior chest wall, the systolic retraction of chronic adhesive pericarditis is altogether more forcible than is this phenomenon.

In cases in which only a limited number of adhesions exist between the epicardium and the visceral pericardium, causing little or no embarrassment of the action of the heart, a diagnosis is extremely difficult.

HYDROPERICARDIUM (HYDROPS PERICARDII)

Clinical Pathology.—Hydropericardium, the presence of serous fluid in the pericardial sac, is usually a sequence of the general anasarca of nephritis or of valvular heart disease. In rarer instances such a serous transudation is the sequence of thrombosis of the cardiac veins. In hydropericardium the pericardial sac contains a variable amount of clear, serous, noninflammatory fluid.

Physical Signs.—The physical signs of hydropericardium are essentially those of fluid in the pericardial sac. There is, however, absence of the friction sound, pain, and fever, which serves to exclude serofibrinous pericarditis. Coupled with a history of cardiac or renal disease, the physical signs point toward hydropericardium, though in many instances the transudation of fluid is but moderate in degree and the condition is entirely overlooked.

HEMOPERICARDIUM

Clinical Pathology.—The accumulation of blood or sanguineous fluid in the pericardial sac occasionally follows stab wounds or penetrating wounds of the pericardium from other causes. It may be due to rupture of the ascending portion of the aorta, which is enveloped by the pericardium, in which event the disease is rapidly fatal, or it may follow rupture of a coronary artery. Occasionally the fluid of serofibrinous pericarditis is tinged with blood.

Physical Signs.—The physical signs of hemopericardium are those of effusion into the pericardium. Added to these signs in certain cases are signs of internal hemorrhage, as pallor, rapid weak pulse, dyspnea, and collapse. In grave cases death ensues early from pressure upon the heart.

PNEUMOPERICARDIUM (HYDRO-, HEMO-, OR PYO-PNEUMOPERICARDIUM)

Clinical Pathology.—Pneumopericardium, the presence of gas in the pericardial sac, is usually associated with the presence of serous fluid (hydro-pneumopericardium), blood (hemo-pneumopericardium), or pus (pyo-pneumopericardium). The disease is but rarely encountered.

Pneumopericardium may result from penetrating wounds of the pericardium; from perforation of the pericardium by a tuberculous cavity of the lung, gangrene of the lung, or pneumothorax; or it may be spontaneous, owing to the development of the bacillus aerogenes capsulatus of Welch. Malignant disease of the esophagus or stomach very rarely causes perforation of the pericardium and consequent pneumopericardium.

Physical Signs.—The disease is occasionally attended by precordial bulging, with absence of the visible cardiac impulse. A pericardial friction sound is occasionally audible and in other instances succussion fremitus is demonstrable. Percussion of the normal area of cardiac dullness in pneumopericardium yields hyperresonance or tympany, while below the level of the fluid in the pericardium the percussion note is dull or flat.

The heart sounds are feeble, being obscured by the pericardial succussion sound, which is a churning sound that has been compared to the sound produced by a water wheel in motion. Occasionally a pericardial friction sound is audible.

Diagnosis.—The pericardial succussion sound is pathognomonic of the disease, and its demonstration renders the diagnosis positive. Pneumopericardium is apt to be confused with left-sided pneumothorax or gaseous distention of the stomach. In left pneumothorax the area of cardiac dullness is not obliterated, and the cardiac impulse is usually visible, though frequently displaced toward the opposite side of the thorax. On the other hand, the tympanitic note of a distended stomach disappears when a stomach tube is passed and the gas is liberated from this viscus.

CHAPTER XIX

DISEASES OF THE ENDOCARDIUM AND VALVES

ACUTE ENDOCARDITIS

Clinical Pathology.—Acute endocarditis occurs in two forms; namely, as *simple acute endocarditis,* and as *malignant* or *infective endocarditis.*

Acute simple endocarditis is almost invariably secondary to disease elsewhere in the bodily economy. It is associated with the greatest frequency with acute rheumatic fever; while next in frequency comes acute tonsillitis, which is followed by scarlatina, and chorea. Any of the acute infectious diseases is liable to be complicated by acute endocarditis. Similarly, certain chronic wasting diseases, as carcinoma, diabetes, and chronic nephritis, are occasionally associated with acute endocarditis.

Recurrent endocarditis is a form of acute endocarditis in which valves which are the site of chronic lesions suddenly "light up" with acute symptoms and signs of endocarditis. Acute endocarditis is noted more frequently in males than in females, and the disease is most prevalent during the third and fourth decades.

The lesions of acute simple endocarditis occasionally affect the endocardium which lines the walls of the chambers of the heart (mural endocarditis); but this number of cases is so small as to be negligible. In the vast majority of cases the changes are in the valves themselves (valvular endocarditis).

The earliest change from the normal in the valve segments is noted at a point a short distance from the free edge, the point where the segments come into apposition when the valve closes. The primary change is a decrease in the size of the endothelial cells with a tendency to assume a cuboidal form, leaving crevices between the individual cells, a state which predisposes to infection of the stroma of the segments. In the further evolution of the disease fibrin is deposited upon the partially denuded areas in consecutive strata, finally forming in many instances excrescences upon the valve cusps, the verrucose type of simple endocarditis. In the event that fragments of these verrucoses are swept away by the circulating blood, they may serve as emboli and lead to thrombosis in the small capillaries of the lungs, brain, or an abdominal organ.

In the further evolution of the disease the verrucose excrescences upon the cardiac valves are prone to organization with the production of scar tissue, which by subsequent contraction causes permanent deformity of the valve in stenosis or incompetence.

Malignant or infective endocarditis, although occurring occasionally as a primary infective inflammation of the endocardium, is in the vast majority of cases secondary to infective disease in more remote portions of the body, developing during the course of puerperal sepsis, osteomyelitis, acute rheumatic fever, or is secondary to erysipelas or acute gonorrhea. Not infrequently malignant endocarditis attacks cardiac valves which are the site of chronic endocardial inflammation.

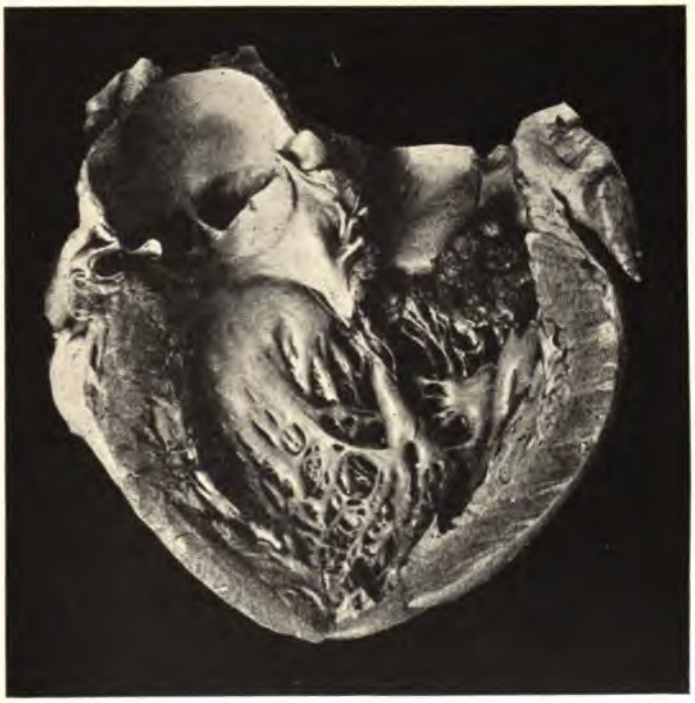


Fig. 176.—Endocarditis, verrucose form. (From Delafield and Prudden.)

In this form of acute endocarditis purulent collections are formed in the connective tissue stroma of the valve segments, the vessels dilate and new vessels from the adjacent myocardium invade the valve stroma. There is a tendency toward repair by sclerosis; but the diseased surface of the segment does not heal with a smooth endothelial surface, but with irregular, villous processes and excrescences, leaving the valves incompetent or stenotic. Perforation of a segment is not infrequent. Infectious emboli are apt to be carried away from the diseased valves by the circulating blood and to initiate metastatic abscesses in distant organs of the body.

Physical Signs.—*Inspection.*—In acute simple endocarditis inspection may yield no physical signs; but in cases of recurrent endocarditis, in which an acute exacerbation of a chronic endocarditis is in progress, there are apt to be in evidence signs of moderate cardiac hypertrophy; namely, displacement of the apex beat, an increase in the force and in the extent of the cardiac impulse, and frequently systolic pulsation of the carotid arteries. In malignant endocarditis, if cardiac dilatation is imminent, the cardiac impulse is feeble and rather diffuse, and is displaced from its normal location in the fifth intercostal space.

Palpation.—The findings upon palpation in acute endocarditis are variable, varying with the duration of the disease and with the form of the disease which is under examination. In recurrent endocarditis the impulse is forcible and heaving, as a rule, as the result of cardiac hypertrophy, and is displaced from its normal site. Not infrequently in this type of the disease a systolic thrill may be defined at one or more of the valve areas. In simple endocarditis in general the cardiac impulse becomes progressively more feeble as the disease progresses; and in malignant endocarditis when cardiac dilatation is imminent the impulse is commonly slapping and weak upon palpation of the precordia.

Percussion.—During the early stage of the disease the area of cardiac dullness is normal; but in recurrent endocarditis it is commonly extended in one or more directions, most frequently toward the left and downward, as a result of cardiac hypertrophy.

Auscultation.—In both simple and malignant endocarditis auscultation frequently elicits a systolic murmur at the mitral or aortic valve area; but this finding does not constitute a pathognomonic sign of acute endocarditis. However, when the murmur develops upon a slightly prolonged or roughened first sound of the heart, indicating coincident stenosis, endocarditis is suggested. In the recurrent form of the disease the murmurs of preexisting valvular disease are audible; and if it is possible to detect changes in the quality of these murmurs in daily examinations, it is probable that an acute endocarditis has developed upon a previous valvular lesion. The pulmonic second sound is commonly accentuated.

Diagnosis.—In the diagnosis of acute simple endocarditis the history of the case is very important. A history of rheumatic fever, acute tonsillitis, or other acute infectious disease, coupled with the meager physical findings, may suggest a diagnosis. Of the physical signs the most important is a systolic murmur at the mitral area, particularly if this develops upon a prolonged and roughened first sound of the heart. Of course, the murmur must be distinguished from a functional murmur; but whereas the murmur of acute endocarditis is most frequently audible in the mitral area, functional murmurs are most commonly elicited at the pulmonic area. Moreover, a functional murmur very rarely involves the aortic valve, over which a murmur is apt to be audible in acute endocarditis.

In the diagnosis of malignant endocarditis regurgitant diastolic murmurs are suggestive in a measure, as functional murmurs are systolic in time. When, moreover, there is a history of previous septic infection and evidence of metastatic infection in other portions of the body, the diagnosis is still more probable. Moreover, malignant endocarditis is attended by fever of a septic type, with precordial distress, and leucocytosis. However, it should be remembered that malignant endocarditis presents a varied symptomatology, sometimes occurring in a cardiac form in which the murmurs of chronic valvular lesions predominate the picture; in a pyemic form with symptoms primarily of metastatic involvement; in a typhoid form, which closely simulates the course of typhoid fever; and finally in a type in which cerebral symptoms as delirium or coma, predominate the picture.

Differential Diagnosis.—From *typhoid fever* it is differentiated by the more abrupt onset of endocarditis, the absence of the step ladder ascent of the fever during the first week, and the presence of precordial distress and dyspnea with chills and leucocytosis, which is in marked contrast to the leucopenia of typhoid fever.

CHRONIC ENDOCARDITIS

Clinical Pathology.—Chronic endocarditis is usually secondary to acute endocarditis, particularly to that form occurring in association with acute rheumatic fever and acute tonsillitis. Other cases of chronic endocarditis arise in persons who have not previously had acute endocarditis, but arise as the result of the constant circulation in the blood of the toxins of lead, alcohol, syphilis, gout, and diabetes. Laborious occupation may initiate the sclerotic process in the aortic segments, as also may arteriosclerosis and chronic nephritis by raising blood pres general circulation.

The changes in the valves consist of a progressiv frequently with the ultimate deposition of calcium valves are thickened, inelastic, and their free border ally coalesce, producing a condition of permanent ste competence. Moreover, in many cases the chordæ te

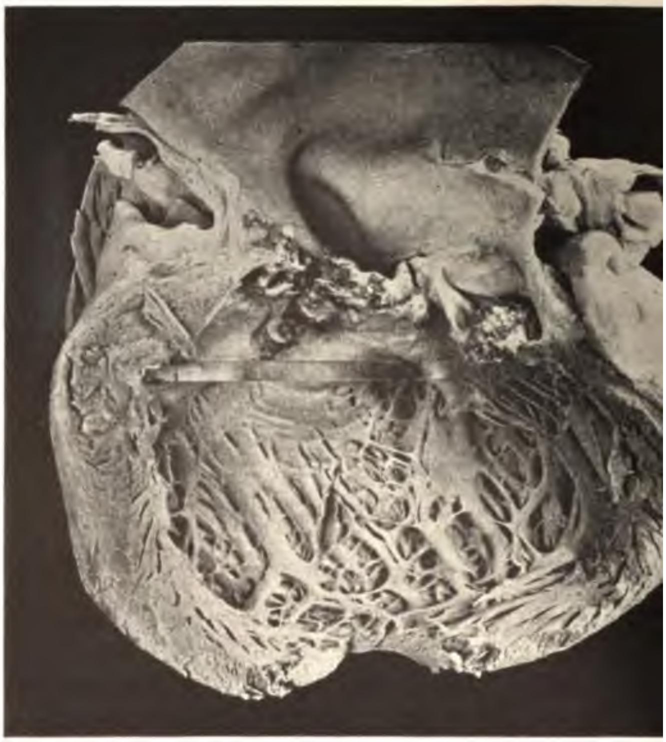


Fig. 177.—Chronic endocarditis. (Delafield and Prudden.)

shortened and thickened so that they no longer perm coaptation of the free borders of the valve segments.

The myocardium of the chamber of the heart upon burden is thrown by the stenotic or incompetent va trophies, to be followed ultimately by myocardial de and cardiac dilatation.

Physical Signs.—The physical signs of chronic endoc essentially those of chronic valvular disease, varyin clinical manifestations with the valve or valves involv

CHRONIC VALVULAR DISEASE

Chronic valvular lesions of the heart are of two types: *stenotic,* which are produced by narrowing of the orifice, offering an obstacle to the onward flow of the blood stream; and *regurgitant,* which are produced by the inability of the valve cusps to prevent the regurgitation of the blood stream as a result of deformity of the valve cusps, shortening of the chordae tendineae, or temporary relaxation of the valvular ring with the result that the normal segments are incapable of closing the abnormally large orifice (relative insufficiency).

Valvular lesions occur at the several valves of the heart in the following order of frequency: (1) mitral, (2) aortic, (3) mitral and tricuspid, (4) mitral and aortic, (5) pulmonary, and (6) tricuspid. Thus, it is noted that isolated organic lesions are encountered with far greater frequency in the valves of the left side of the heart than at the pulmonary and tricuspid valves. Organic lesions affecting the valves of the right side of the heart are, in the vast majority of cases, of congenital origin; but regurgitant lesions also occur in this portion of the valvular mechanism as the result of left-sided lesions of extensive duration. Also, in rare instances endocarditis attacks these valves coincidentally with the aortic and mitral valves.

Organic lesions of the mitral valve are usually the sequence of acute endocarditis, while lesions of the aortic valve may be of endocarditic origin, or they may result from general arteriosclerosis.

Effects of Valvular Lesions.—The mechanical effects of valvular lesions are classified as *primary,* as they are exerted upon the myocardium; and as *secondary,* as they are expended upon more remote organs and tissues of the body.

The natural tendency of a valvular lesion is to diminish the quantity of circulating blood in advance of the lesion, and to increase the quantity of blood in the circulatory system behind the site of the lesion; hence, to lower arterial tension and to raise venous pressure. In the presence of a stenotic lesion the chamber discharging blood through the narrowed orifice can only discharge a portion of its contents during systole, while during diastole the chamber receives a certain physiologic quota of blood from the source which discharges into it. Similarly, in the case of a regurgitant lesion, at each systole the incompetent valve permits a certain portion of the contents of the chamber in advance of the lesion to regurgitate into the chamber behind it, which coincidentally is receiving its physiologic quota from its source of supply. In this manner an imbalance is induced in the circulation, the tendency of which is to produce a diminution of arterial tension and a corresponding increase in venous pressure.

If there were no means of balancing the disproportion between the pressures in the arterial and venous circulations, death would supervene early upon the establishment of a valvular lesion of the heart. But to offset the difference in pressure in the two systems, the chamber of the heart upon which the increased burden is thrown compensates for the incompetence of the valve by hypertrophy of the myocardium, and the imbalance of the circulation is corrected for a variable time. As long as this compensation is maintained, the valvular lesion is unattended by subjective symptoms; but when the reserve power of the myocardium is exhausted and the heart fails to adequately expel its contents, compensation is said to be broken; and signs of cardiac insufficiency rapidly supervene.

The effects of valvular lesions are well illustrated by the sequence of events following regurgitation at the aortic valve. When this valve is incompetent a variable quantity of the blood which is expelled into the aorta during ventricular systole regurgitates into the left ventricle during diastole. An increased burden is then thrown upon this chamber of the heart, which is at the same time receiving its quota of blood from the left auricle during ventricular diastole; and the left ventricle undergoes a transient dilatation, the physiological "active or compensatory dilatation" of Krehl.

In response to the increased demand for work, however, the left ventricle hypertrophies, and the imbalance between the arterial and venous circulations is restored. As long as this hypertrophy is maintained, no change is noted in the general circulation; but the time ultimately arrives in organic disease of the valve when the ventricular musculature is no longer able to sustain the extra burden, and permanent dilatation of the left ventricle, the "relaxation dilatation" of Sahli, supervenes.

The dilatation of the left ventricle is attended by stretching of the muscular ring at the base of the mitral valve, and the normal cusps of this valve are unable to adequately close the orifice, with the result that a variable quantity of blood regurgitates into the left auricle during ventricular systole, the period of the cardiac cycle during which the auricle is receiving blood from the lungs. Under the influence of this double supply and increased load, the left auricle undergoes a transient dilatation, to ultimately hypertrophy in compensation for the valvular insufficiency.

After a variable period this compensatory hypertrophy of the auricle yields to dilatation whereupon, by virtue of the regurgitation of blood from the left ventricle, which the dilated auricle is unable to expel during systole, the blood pressure in the pulmonary circulation is raised, as evinced by accentuation of the pulmonary second sound.

The continuous engorgement of the pulmonary circulation predisposes to the development of catarrhal inflammation of the bronchi, and, in more extreme grades, to edema of the lung or hydrothorax.

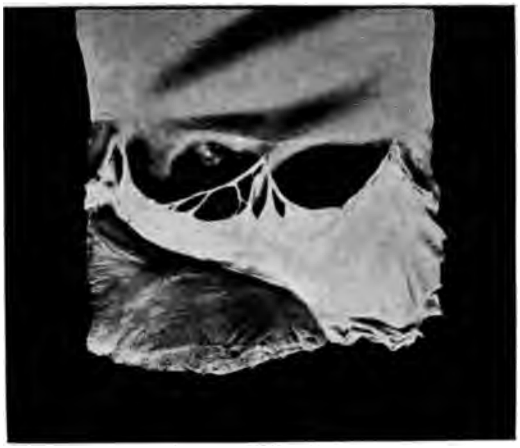


Fig. 178.—Fenestration of semilunar valves. (From Delafield and Prudden.)

The heightened tension in the pulmonary circuit is attended by coincident hypertrophy or transient dilatation and subsequent hypertrophy of the right ventricle, compensating for a time the insufficiency of the left heart. But the right ventricle finally yields to dilatation, with the induction of relative tricuspid regurgitation and engorgement of the right auricle.

The right auricle, which is not capable of any great degree of compensatory hypertrophy, early suffers dilatation, attended by general venous stasis with systolic pulsation of the liver and effusion of fluid into the serous cavities and cellular tissues of the body.

AORTIC REGURGITATION (AORTIC INSUFFICIENCY; AORTIC INCOMPETENCE; CORRIGAN'S DISEASE)

Clinical Pathology.—Accurate and complete closure of the aortic valve is dependent upon accurate coaptation of the free borders of the cusps of the semilunar valve, which are forced together by the arterial pressure in the root of the aorta just subsequent to the completion of ventricular systole. When, as a sequel of recent endocarditis, vegetations are formed upon the valvular cusps, accurate coaptation of the valve segments does not occur during ventricular diastole and a portion of the blood which is expelled from the ventricle during its systole is allowed to regurgitate into the ventricular chamber during diastole. Similarly, shrinking and sclerosis of the segments occurring as a result of chronic endocarditis prevents accurate coaptation of the cusps with the induction of regurgitation at the aortic orifice. In this class of cases there are frequently adhesions between the individual cusps. In yet other instances there is fenestration of one or more cusps, arising as a congenital defect, or as the result of ulcerative endocarditis. But there is another group of cases in which, without any disease or deformity of the valvular cusps, the fibrous ring at the aortic orifice is enlarged with the result that the normal valvular cusps are no longer capable of closing the abnormally large aortic orifice, resulting in the induction of *relative aortic regurgitation.* This change is encountered in con-

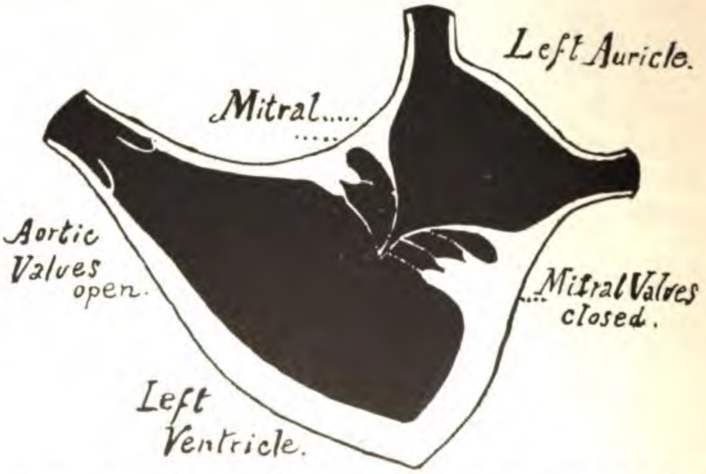


Fig. 179.—Normal ventricular systole. Mitral valve is closed; aortic valve is open.

nection with syphilitic aortitis and aneurysm of the ascending portion of the aorta. In yet another group of cases aortic regurgitation develops as the consequence of a slow sclerosis of the valvular segments and aortic ring, occurring as part and parcel of generalized arterial sclerosis in middle-aged male subjects who have for years followed strenuous occupations. Mineral intoxication, particularly plumbism, is the initiating cause in certain cases, whereas other cases develop in subjects of a gouty diathesis. Aortic regurgitation is prone to develop during middle life, although isolated cases are picked up in younger subjects; and the vast majority of the cases occur in the male sex.

The mechanical influences of the lesion in aortic regurgitation are classified as *primary*, as they are exerted upon the myocardium, and as *secondary*, as they are expended upon more remote organs and tissues of the body. As a result of the progressive and consecutive manifestation of these influences, three distinct stages or periods of the disease may be diagnosed and employed by the examiner as a basis for his prognosis and treatment of the disease.

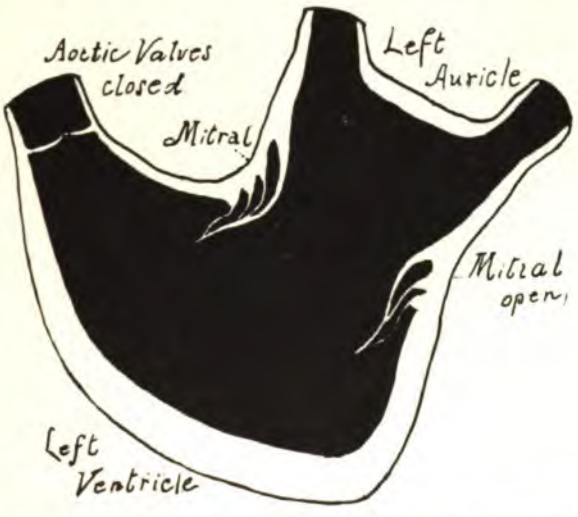


Fig. 180.—Normal ventricular diastole. Mitral valve is open. Aortic valve is closed.

During the first stage of the disease the brunt of the lesion falls upon the left side of the heart and the arterial system, and the myocardial changes are limited to the left auricle and left ventricle. During the second stage of the disease, with the gradual failure of the left heart, the pressure rises in the pulmonary circulation and the lesion is compensated by hypertrophy of the right ventricle.

Finally, the third and terminal stage of the disease is ushered in with failure of right ventricular hypertrophy and engorgement of the general venous circulation.

During the *first stage* of the disease the primary effect of the aortic lesion is exerted upon the left ventricle during its diastole, at which period it becomes the target for two streams of blood entering its chamber simultaneously, the one propelled into the ventricle by left auricular systole, the other regurgitating into the ventricle from the aorta by reason of the incompetent semilunar valves. Under the stress of this added burden the left ventricle undergoes a transient dilatation which is compensated by hypertrophy of the myocardium of this chamber of the heart. So long as this compensatory hypertrophy is maintained, the ventricle is capable of expelling its contents during ventricular systole and no added burden is thrown upon the pulmonary circulation.

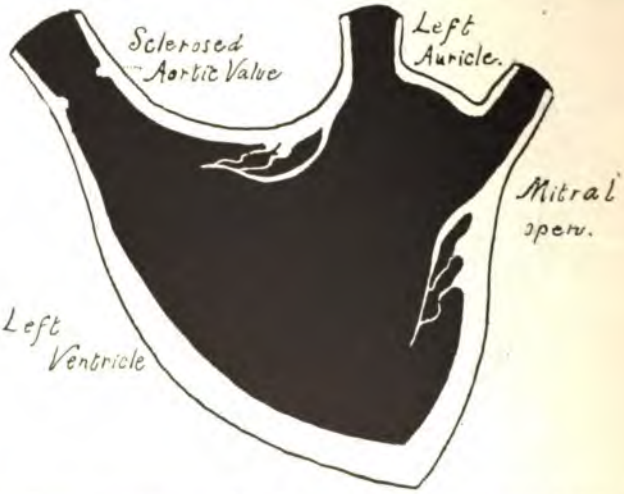


Fig. 181.—Aortic regurgitation. Mitral and aortic valves are open during diastole.

Upon the general arterial circulation, however, the regurgitant lesion exercises a decided influence. At each ventricular systole an excessive quantity of blood is expelled into the aorta, and this quota of blood is expelled with undue force by the hypertrophied ventricle. In this manner the arterial system is abruptly surcharged with blood during ventricular systole, resulting in an abrupt rise in arterial pressure, which is almost immediately followed by as abrupt a fall during ventricular diastole. The effect of the sudden expulsion of the ventricular contents is to distend the arteries which undergo a transient dilatation, with subsequent collapse, producing a pulse wave which not infrequently extends into the capillaries as the capillary pulse of Quincke, and which is occasionally demonstrable in the superficial veins as the centripetal venous pulse. The strain which is thus thrown upon the aorta and general arterial system tends to provoke generalized arterial sclerosis in which the coronary arteries share, with consequent myocardial degeneration.

As the disease progresses, the left ventricle hypertrophies to an excessive degree in its effort to supply the tissues of the body with their normal quota of arterial blood, resulting in the *cor bovinum* of this disease. During the first stage of the disease the subject of the malady is troubled with recurrent transient failures of compensation which are compensated by subsequent restoration of compensation by the left ventricle under repose; but the time eventually arrives when through myocardial weakness the ventricle undergoes dilatation beyond the power of compensation and the mitral valve yields with the establishment of relative mitral regurgitation. In other instances the mitral valve is attacked by endocarditic changes occurring as a result of the continued hypertension in the left ventricle, but the usual mechanism of the establishment of mitral insufficiency is through dilatation of the ventricle.

With the establishment of mitral regurgitation the *second stage* of the disease is ushered in with embarrassment of the pulmonary circulation and compensation by hypertrophy of the right ventricle, as evinced by accentuation of the second sound of the heart at the pulmonic area. In the vast majority of cases the establishment of relative mitral insufficiency during the course of aortic regurgitation is to be considered a distinctly benign complication of the disease, and one which is compensated by right ventricular hypertrophy over a prolonged period of time. In most cases in which relative mitral insufficiency develops, it occurs as a signal that the compensatory hypertrophy of the left ventricle is unable to keep pace with the tendency toward dilatation from nutritional changes in the myocardium, and may be likened to the safety-valve leak of tricuspid insufficiency which for a time relieves the strain thrown upon a laboring right heart later in the course of the disease.

During the second stage of the disease bronchial inflammation is frequently an annoying feature of the case, the intensity of the symptoms and signs varying with the reserve power of the right ventricle.

Compensation by right ventricular hypertrophy is maintained for a variable period in aortic regurgitation; but with the continued hypertension existing in the pulmonary circulation the reserve power of the right heart is overtaxed, and with the supervention of right ventricular failure the third stage of the disease ensues. The right ventricle under these circumstances fails through one of two mechanisms. The tricuspid valve, in the presence of the progressive increase in intraventricular pressure may yield and permit a portion of the contents of the ventricle to regurgitate into the right auricle during ventricular systole in the absence of any definite yielding of the right ventricular wall, with the establishment of the so-called ''safety-valve leak'' of the tricuspid orifice. If, on the contrary, the tricuspid valve retains its integrity in the presence of the progressive rise in the intraventricular pressure, the point is attained when the right ventricle is no longer capable of surmounting the excessive pressure in the pulmonary artery, and the ventricle is only partially emptied during ventricular systole. To the residual blood in the right ventricle is added the contents of the right auricle at the succeeding auricular systole, and the walls of the surcharged right ventricle yield, with dilatation of the ventricle and permanent enlargement of the auriculo-ventricular orifice. Thus is induced the terminal primary effect of the regurgitant aortic lesion, with the establishment of incompetence of the tricuspid valve with dilatation of the right ventricle.

During this late stage of the disease the secondary effects of the aortic lesion develop in rapid succession. The venous return from the trunk and extremities, pouring into the right auricle from the venæ cavæ, encounters a distinct resistence in the regurgitant lesion at the tricuspid valve. The ultimate result is a marked diminution of the volume of blood which reaches the arterial circulation and a dangerous increase of the volume of blood contained in the venous system. As a diminished volume of blood is expelled at each systole of the right ventricle, the left heart is only partially filled, the left ventricle contracts upon a markedly diminished quota of blood and there is a progressive fall in arterial tension. On the other hand, as a result of the progressive stasis in the venous system, passive congestion of the lungs and abdominal viscera develops, together with effusion into the pleura and peritoneum, and the extravasation of fluid into the cellular tissues of the body.

Aortic regurgitation is not infrequently associated with stenosis of the valve, particularly in cases of endocarditic origin. In these cases the lesion which prevents accurate coaptation of the valvular cusps during ventricular diastole likewise produces narrowing of the orifice during ventricular systole.

In the first and second stages of the disease the subject of aortic regurgitation does not present many symptoms unless he be subjected to sudden strain. There is moderate dyspnea upon exertion, and occasionally considerable nervous irritability. With the failure of compensation by the right ventricle, bronchial symptoms come to the fore and the subject complains of digestive disturbances, frequently of considerable duration. Dizziness frequently occurs from cerebral anemia, and, indeed, sudden death may result from embolism of the brain. Coronary disease may induce attacks of angina, and as the disease progresses dyspnea gives place to orthopnea, with excessive cough and the raising of blood-tinged expectoration.

Physical Signs.—*Inspection.*—In the first stage of aortic regurgitation the cardiac impulse is forceful; its area is more extensive than in the case of the impulse of the normal heart; and the impulse is displaced toward the left and downward as a result of left ventricular hypertrophy. The impulse is observed to be heaving and slightly prolonged, and frequently the area of impulse is surrounded by a circumscribed area of systolic retraction of the thorax. As left ventricular hypertrophy progresses, the apex beat is observed to occupy a site farther and farther from the median line of the thorax. A very characteristic sign of the disease is systolic pulsation visible over the carotids in the lower cervical region, and frequently there are visible pulsations of the subclavian and the brachial arteries. The capillary pulse of Quincke is demonstrable in many cases of the disease and in a smaller number, a centripetal venous pulse is to be detected.

In the second stage of aortic regurgitation there is a striking diminution in the intensity of the signs emanating from the hypertrophied left ventricle as hypertrophy gives place to dilatation. The cardiac impulse becomes less forceful and more diffuse and undulatory, while at the same time being carried farther from the median line and downward. The systolic pulsations of the arteries decrease in force and eventually disappear. The respiratory movements of the thorax now become more frequent and the facies is anxious. Systolic pulsation of the epigastrium is commonly encountered from right ventricular hypertrophy.

During the third stage of the disease the cardiac impulse is very diffuse, feeble, and undulatory, occupying a broad area along the left sternal border between the third and sixth interspaces. The positive venous pulse appears in the jugulars with the establishment of tricuspid insufficiency, and this same state is frequently attended by a systolic pulsation of the liver. The subject suffers with dyspnea and harassing cough, and is no longer able to assume the dorsal decubitus.

Palpation.—In the first stage of the disease with the maintenance of left ventricular hypertrophy the cardiac impulse is heaving and forceful, with great lifting power, and is frequently immediately followed by a moderate retraction of the thoracic wall in the vicinity of the impulse. As left ventricular dilatation becomes imminent the apex beat becomes appreciably enfeebled and rather slapping than thrusting as in the first instance. A diastolic thrill is occasionally to be detected at the aortic area, though a thrill is not present with aortic regurgitation with the same constancy as in the case of stenosis of this orifice.

In the second stage of the disease, with the establishment of mitral regurgitation and hypertrophy of the right ventricle, the cardiac impulse is perceptible farther toward the median line and downward toward the epigastrium. A distinct systolic pulsation of the upper epigastrium is almost constantly present with hypertrophy of the right ventricle.

With the progressive failure of the right ventricle which ushers in the third stage of aortic regurgitation, the cardiac impulse becomes feeble, diffuse, and undulatory, at the same time extending beyond the right sternal margin and well downward into the epigastrium. The positive venous pulse is demonstrable in the jugular vein by compression of the vessel above the clavicle, and systolic pulsation of the liver can usually be detected by bimanual palpation of the hepatic region.

The *pulse* of aortic regurgitation is of the water-hammer or Corrigan type, with an abrupt distention of the artery which is followed immediately by collapse of the vessel. This character of pulse is readily detected by grasping the forearm just above the wrist and elevating the subject's arm above the level of the heart, whereupon the force of gravity facilitates the emptying of the artery during ventricular diastole. With the inception of mitral regurgitation in the second stage of the disease the volume of the radial pulse is diminished and its rhythm is disturbed; and with the failure of the right ventricle the pulse is feeble, rapid and running, and is totally arrhythmic.

Percussion.—Percussion of the anterior surface of the thorax in aortic regurgitation gives an outline of cardiac dullness which varies directly with the changes which occur in the size and shape of the chambers of the heart. During the first stage of the disease the transverse dullness of the heart is extended to the left and downward as a result of left ventricular hypertrophy. At this period of the disease the apical area is quite discrete and pointed, to become rounded and obtuse with the establishment of right ventricular hypertrophy in the second stage of the disease. At this time the transverse dullness of the heart is increased toward the right of the sternum, where it encroaches upon the vesicular resonance of Ebstein's cardiohepatic angle, and is extended downward into the upper epigastrium. With the supervention of dilatation of the right ventricle the cardiac outline extends further to the right of the sternum and downward into the upper portion of the epigastrium. In cases which are associated with aneurysm of the ascending aorta, percussion of the area of vascular dullness at the base of the heart yields an extension of dullness to the right of the sternum in this region.

Percussion of the pulmonary bases during the first and second periods of the disease yields little, if any, alteration in the character of the percussion sounds elicited. In the third stage of the disease, however, with the establishment of pulmonary edema, there is dullness over the bases posteriorly; and, in the presence of hydrothorax, there is frank flatness over the distribution of the effusion. Similarly, during the terminal stage of the disease the areas of hepatic and splenic dullness are found to be extended, as a result of chronic passive congestion of these organs.

Auscultation.—In aortic regurgitation there is generated a murmur with its point of maximum intensity at the aortic valve area near the junction of the second right costal cartilage with the sternum, or over the middle of the gladiolus, or near the apex of the heart. The point of maximum intensity of this murmur is thus variable; but in the majority of instances it will be localized in the aortic area in the second right interspace. The line of transmission of the murmur passes in the majority of cases downward along the right sternal margin toward the ensiform cartilage, though occasionally it crosses the median portion of the sternum and is conducted to the apex of the heart. The murmur usually replaces the second sound of the heart at the aortic area, but in certain cases both murmur and second sound are audible.

The quality of the murmur is variable during the different stages of the disease, and its quality and intensity are also dependent upon the character of the underlying lesion and upon the degree of regurgitation. During the first stage of the disease with the maintenance of left ventricular hypertrophy with its excessive output of blood at each ventricular systole the murmur is commonly loud and blowing, but seldom as harsh as is the murmur of aortic stenosis. The narrower the orifice through which the blood stream regurgitates, the harsher is the murmur. Similarly a prolongation of the murmur points to a narrow orifice at the incompetent valve. A brief "whiff" at this area, on the contrary, in which the murmur occupies only the early portion of diastole and is quickly attenuated and disappears, points to a wide orifice with the regurgitation of a more considerable quantity of blood.

With dilatation of the left ventricle and the establishment of relative mitral regurgitation during the second stage of the disease the murmur at the aortic area loses much of its intensity, and there is added a soft, blowing systolic murmur at the mitral area, which is transmitted downward and toward the left axilla, and is attended by accentuation of the pulmonic second sound. There is occasionally generated at the mitral area the murmur of Austin Flint, the mechanism of which has been described in a previous section.

With the supervention of right ventricular failure in the third stage of the disease, the murmurs at the aortic and mitral areas become feeble or are entirely inaudible; the formerly accentuated pulmonic second sound gives place to an enfeeblement of this sound; and the systolic murmur of tricuspid regurgitation becomes audible in the tricuspid area.

In every case of suspected aortic regurgitation the examiner should endeavor to pick up a transmitted diastolic murmur over the carotid arteries. During the first stage of the disease this murmur is usually quite distinctly audible, to become enfeebled or lost during the second and third stages of the disease. Similarly, auscultation of the femoral artery will occasionally elicit the double murmur of Duroziez in aortic regurgitation.

As aortic regurgitation and stenosis not infrequently coexist, the examiner will occasionally detect a double "see-saw murmur" at the aortic area, the one systolic and harsh in quality, the other diastolic and more musical and less intense.

Diagnosis.—The diagnosis of aortic regurgitation is as a rule readily made upon the physical findings which are presented. The presence of a diastolic murmur with its point of maximum intensely localized in the aortic area and transmitted downward and toward the left, the signs of immense hypertrophy or dilatation of the left ventricle, pulsations in the carotid arteries, the water-hammer pulse, and Duroziez's double femoral murmur, are the cardinal signs upon which the diagnosis is based.

AORTIC STENOSIS

Clinical Pathology.—Simple stenosis of the aortic valve is the least frequently encountered of all acquired valvular lesions of the heart, and the cases occur more frequently as part and parcel of general arteriosclerosis in middle-aged subjects than as a sequela of acute endocarditis attacking the valvular segments. Thus, in the majority of instances, the valvular change consists of a gradual and progressive sclerosis of the cusps and sclerotic contraction of the aortic ring, resulting in diminution in the caliber of the aortic orifice. Aortic stenosis occasionally arises as a result of congenital malformation of the valve, in which event the lesion may consist of a fusion of the cusps with a mere slit or chink between the free borders of the leaflets. In yet other cases the valve segments are the site of numerous excrescences which project into the lumen of the orifice during ventricular systole with the induction of narrowing of the physiologic orifice of the valve at this period of the cardiac cycle.

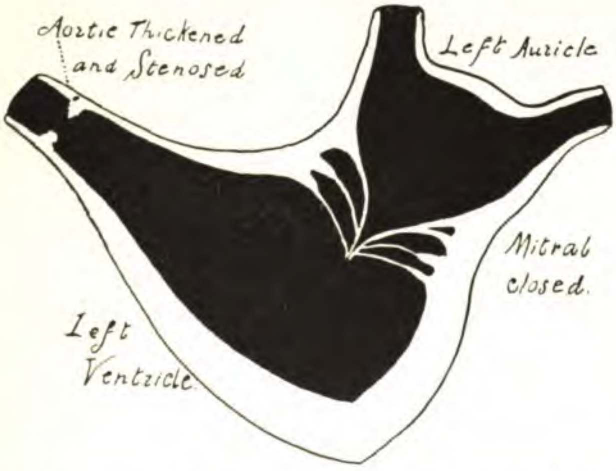


Fig. 182.—Aortic stenosis. Mitral valve is closed during ventricular systole. Aortic orifice is stenotic.

But if organic derangement of the aortic valve is a rare lesion, there are, nevertheless, a number of possible factors which are capable of generating a systolic murmur at this valve in the absence of organic disease of the aortic ring or segments. In the presence of simple endocarditis the segments may bear minute excrescences which, while producing no actual stenosis of the establishment of relative mitral regurgitation. The establishment of the mitral insufficiency relieves the laboring ventricle by a reduction of intraventricular pressure, but it serves to increase the tension of the pulmonary circulation and to throw an added burden upon the right ventricle, which hypertrophies in response to the increased demand for work.

The *second stage* of the disease, which is ushered in with the introduction of relative mitral insufficiency is readily recognized by the accentuation of the pulmonary second sound which ensues upon right ventricular hypertrophy and the increasing hypertension in the pulmonary circulation. The course of the second stage of aortic stenosis pursues the same course as has been detailed in the section upon aortic regurgitation.

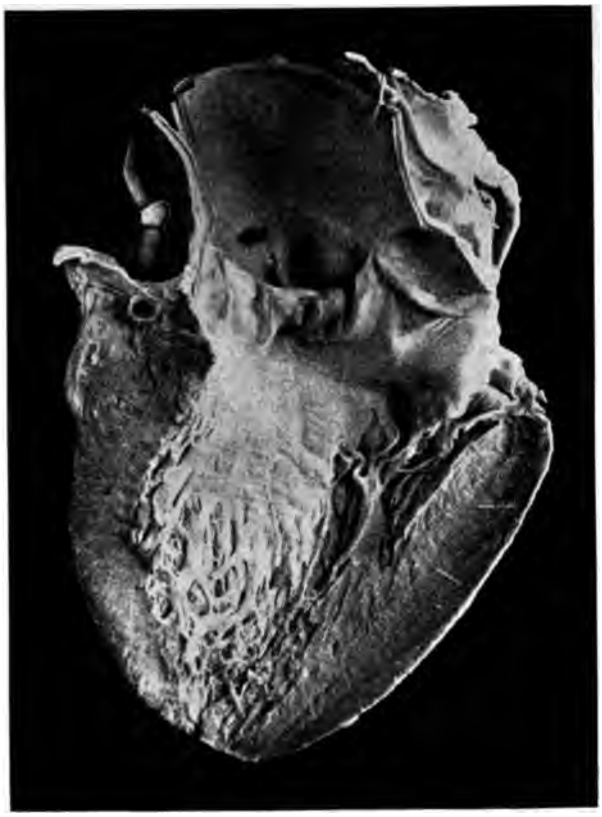


Fig. 183.—Chronic endocarditis with coalescence of two aortic cusps. (From Delafield and Prudden.)

The *third stage* of aortic stenosis is ushered in with failure of stenosis and upon the contractile power of the left ventricle. The more rigid and unyielding is the aortic ring from sclerosis or calcareous deposit, and the greater the hypertrophy of the left ventricle, the more intense is the systolic thrill at the aortic area. With the advent of imminent failure of the left heart there is a progressive diminution in the intensity of the thrill, which finally becomes imperceptible.

When the left ventricle dilates and mitral regurgitation is established during the second stage of the disease the cardiac impulse loses its forceful lifting character, to become feeble, slapping, and undulatory. The valve shock over the pulmonic area becomes accentuated as a result of right ventricular hypertrophy, and palpation of the hepatic region may reveal a pseudopulsation which is due to the transmitted impact against this organ of the right ventricle during its forcible systole.

During the third stage of the disease the cardiac impulse is feeble and undulatory, with irregularity of both force and rhythm. A thrill at the aortic area is not to be encountered at this late stage of the disease, and failure to detect a thrill during the later periods of the disease is not conclusive evidence of the absence of aortic stenosis.

The pulse in aortic stenosis is slow and sustained for an appreciable interval beneath the palpating finger. Its volume is diminished, the diminution varying with the degree of stenosis and the compensation by left ventricular hypertrophy. With the finger upon the radial artery the examiner is instinctively reminded of the slow and forcible systole of the hypertrophied left ventricle, expelling its contents through a narrowed aortic orifice. The sphygmographic tracing of the pulse of aortic stenosis exhibits a prolonged and oblique anacrotic limb, capped by a prolonged plateau, and followed by a slow descent to the base line. During the last stage of the disease the pulse is rapid, diminished in force and volume, and totally disordered in rhythm.

Percussion.—During the first stage of the disease, so long as left ventricular hypertrophy is moderate, little or no alteration may be encountered in the area of cardiac dullness. With the progress of the disease there is a progressive extension of the dullness toward the left axillary line and downward. In the interpretation of percussion findings in this disease the examiner should make due allowance for alveolar distention of emphysema if such be present.

With left ventricular dilatation in the second period of the tion of the cardiac apex will reveal the blowing systolic murmur of relative mitral insufficiency. At the same time the pulmonary second sound at the second left intercostal space is markedly accentuated as a consequence of right ventricular hypertrophy.

With failure of the right heart during the third stage of the disease there is a progressive diminution of the intensity of the aortic systolic murmur, which frequently becomes inaudible late in the disease. At the same time the pulmonary second sound loses its accentuation with the development of the soft blowing safety-valve leak at the tricuspid orifice.

Auscultation of the bases of the lungs now reveals the presence of numerous moist râles, or the respiratory murmur may be abolished over an extensive area in the presence of effusion into the pleural cavity.

Diagnosis.—The detection of a harsh systolic murmur with its point of maximum intensity at the aortic area and transmitted upward into the root of the neck, attended by a palpable thrill, and a slow, forceful, and heaving apex beat which is displaced to the left and downward assures the diagnosis of stenosis at the aortic orifice. In no other disease of the heart do we see a greater contrast than that which is presented in this disease between the powerful and heaving apex beat and the small radial pulse of aortic stenosis.

The murmur of *relative aortic stenosis,* which is caused by dilatation of the aorta distal to the valve, is systolic in time, but it is unattended by alterations of the radial pulse or ventricular hypertrophy; and it is attended by pulsations of the carotid vessels, accentuation of the aortic second sound, and an extension of dullness to the right of the sternum.

Functional murmurs are only occasionally encountered at the aortic area. When present, they are musical, transient, and not transmitted beyond the limits of the precordia. They do not cause alterations in the cardiac outline or the pulse, and are usually encountered in young or anemic subjects.

MITRAL REGURGITATION (MITRAL INSUFFICIENCY; MITRAL INCOMPETENCE)

ical Pathology.—Complete closure or competence of the orifice is dependent upon accurate coaptation of the segf the valve, active traction upon the cusps exerted by the muscles through the medium of the chordæ tendineæ cardial degeneration, the contraction of the ventricle may prove nsufficient to reduce an auriculoventricular orifice which is unduly large, and the chordæ tendineæ may not permit the accurate coaptation of the normal cusps, with the consequent induction of *relative mitral regurgitation.* Stenosis or insufficiency of the aortic valve as well as bodily states which are attended by marked hypertension of the greater circulation, such as general arteriosclerosis, chronic interstitial nephritis, and cirrhosis of the liver, are very productive of regurgitation at the mitral valve.

The mechanical influences of mitral regurgitation are *primary,* as they are exerted upon the myocardium, and *secondary* as they affect more remote organs and tissues of the body. The clinical manifestations of these influences upon the circulatory imbalance which is induced by the mitral lesion vary in direct proportion to the degree of incompetence and with the extent to which the disturbance in function is compensated by hypertrophy of the myocardium.

During the *first stage* of mitral regurgitation the brunt of the lesion is expended upon the left side of the heart. The imperfect coaptation of the mitral cusps permits a variable quantity of blood to regurgitate into the chamber of the left auricle during ventricular systole. At this period of the cardiac cycle, which corresponds to auricular diastole, the left auricle is receiving the discharge of blood which is returned from the lungs by the pulmonary veins. Thus, the auricle becomes the target for two streams of blood, the one entering its chamber from the pulmonary veins, the other regurgitating from the left ventricle. The fusion of the two streams results in the induction of the so-called "fluid veins," with the generation of an endocardial murmur; and under the influence of these factors the surcharged auricle undergoes a transient dilatation, which is followed by compensatory hypertrophy of its wall, whereby it discharges an excess of blood into the left ventricle at each auricular systole. But the mitral insufficiency is still there and not all of this increased auricular output is propelled by the left ventricle into the aorta during its systole. On the contrary, at each ventricular systole a portion of the ventricular blood is returned to the auricle; and to compensate for the extra burden which is thrown upon it, the left ventricle undergoes compensatory hypertrophy.

In this early stage of the disease the organic change is limited to the left side of the heart. During a period which is variable the hypertrophied left auricle is capable of expelling its contents during systole, and during this time there is no embarrassment of the pulmonary circulation and no extra demand is made upon the right ventricle. Similarly, for a time the hypertrophied left ventricle expels approximately the physiologic quantity of blood into the aorta during its systole, and the normal tension of the general circulation is maintained.

After a period varying with the reserve power of the left heart, the second stage of the disease ensues, in which the mechanical influences of the mitral lesion are exerted upon the pulmonary circulation and the right ventricle. The hypertrophied left auricle is now no longer capable of completely expelling its contents during systole and dilatation beyond the power of compensation ensues. The onward progress of the pulmonary circulation is progressively impeded, and a condition of hypertension is established in this circuit, as evinced by accentuation of the pulmonary second sound. The persistent hypertension induces a varying grade of sclerosis of the pulmonary arterial system, which adds further to the burden thrown upon the right ventricle. At the same time chronic catarrhal inflammation of the bronchial mucous membranes is prone to develop.

The right ventricle undergoes compensatory hypertrophy in response to the increased demand for work, a hypertrophy which during a variable period of time is capable of compensating for the damage at the mitral valve, and one which progressively increases up to a point which is dependent upon the integrity of the tricuspid valve and the reserve power of the myocardium. As long as the right and left ventricles, acting in unison, are capable of propelling an adequate quantity of blood through the pulmonary and the general circulations, there is no apparent imbalance between the two systems. But when this equilibrium is disturbed, the right ventricle dilates beyond the power of compensation, and the third stage of the disease is ushered in with failure of the right heart and passive congestion of the general venous system.

With the progressive hypertension in the pulmonary circuit, the right ventricle fails through one of two mechanisms. The tricuspid valve, in the presence of the rising intraventricular pressure may yield and permit a portion of the blood to regur
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pressure in the pulmonary artery. Under these circumstances the ventricle is only partially emptied during ventricular systole. To the residual blood in the right ventricle is added the contents of the right auricle at the succeeding auricular systole; and the walls of the surcharged ventricle yield, with enlargement of the auriculoventricular orifice. In this manner is induced the terminal primary effect of the mitral lesion, with the establishment of incompetence of the tricuspid valve, constituting *relative tricuspid insufficiency.*

At this stage of the disease the secondary effects of the mitral lesion develop in rapid succession. The venous return from the trunk and extremities, pouring into the right auricle from the venæ cavæ, encounters a distinct resistance in the regurgitant lesion at the tricuspid valve. The ultimate result is a marked diminution of the volume of blood which reaches the arterial circulation and a dangerous increase of the volume of blood contained in the venous system. As a diminished volume of blood is expelled at each systole of the right ventricle, the left ventricle is only partially filled during diastole, and there is a progressive fall in the general arterial tension. On the other hand, as a consequence of the progressive stasis in the venous system, passive congestion of the lungs and abdominal viscera develops together with effusion into the serous sacs and cellular tissues of the body.

Mitral regurgitation is not infrequently associated with stenosis of the valve, particularly in cases of endocarditic origin. In these cases the lesion which narrows the auriculoventricular orifice likewise prevents accurate coaptation of the cusps during ventricular systole.

Regurgitation at the mitral valve is attended by few symptoms in the first stage when compensation is as yet unbroken. Physical exertion is usually followed by moderate shortness of breath or a fit of cough with the expectoration of a little frothy sputum, which occasionally is tinged with blood. In the second and third stages of the disease, owing to the embarrassment of the pulmonary circulation, symptoms of bronchial inflammation develop with various grades of dyspnea, which finally eventuates in frank orthopnea. Cough is now a constant accompaniment of the disease. Expectoration is profuse, the sputum containing numerous desquamated alveolar epithelial cells which contain blood pigment, the so-called "heart-failure cells."

The subject of advanced mitral regurgitation is subject to disturbances of digestion, and with the progressive venous stasis the

the sixth intercostal spaces, and frequently also in the epigastrium along the left costal arch. At this time also the positive venous pulse is demonstrable in the jugular veins as a result of tricuspid leakage, and occasionally there is a systolic hepatic pulsation arising as a result of the same lesion.

At the same time signs of general venous stasis appear, as evinced by edema of the feet and ankles, progressive abdominal enlargement from ascites, chronic cough with the raising of serous, sometimes blood-streaked expectoration, together with acceleration of the respiratory movements of the thorax and cyanosis of the mucous membranes, auricles, and digits. In chronic cases of extensive duration clubbing of the fingers is frequently seen, and these subjects often exhibit a tortuosity and distention of the superficial veins of the neck and chest.

Palpation.—During the maintenance of left ventricular hypertrophy in the first stage of the disease the cardiac impulse is discrete, forceful, and heaving upon palpation of the precordia. In a relatively small percentage of cases of pure mitral regurgitation a fine systolic thrill is to be detected over the cardiac apex. A thrill is, however, much more commonly encountered in the cases of combined stenosis and regurgitation at the mitral orifice.

In the second stage of the disease, with the development of compensatory hypertrophy of the right ventricle, the palpable impulse of the heart becomes more extensive, while preserving its force. Systolic lifting of the lower costal margin and upper epigastrium is frequently to be detected, as well as a systolic shock communicated to the liver by the overacting right ventricle.

With the progressive failure of the right ventricle during the third stage of the disease the cardiac impulse becomes progressively enfeebled, diffuse, and wavy, frequently extending to the right of the sternum and well into the epigastrium. Positive systolic pulsation of the jugular veins is to be detected by compression of the vein just above the clavicle, a simple maneuver which abolishes a true systolic jugular pulsation and thus serves to distinguish it from a false pulsation communicated to the vein from the subjacent carotid artery.

The *pulse* in mitral regurgitation is to a large degree indicative of the state of the left ventricle throughout the course of the disease. So long as compensation is maintained, the radial pulse is of constant volume and tension and its rhythm is usually little disturbed. Moderate acceleration is frequently present, however, and there exists a small group of cases in which arrhythmia is a striking feature throughout the course of the disease. With the supervention of left ventricular dilatation the volume of the pulse is markedly reduced, acceleration is more marked, and irregularity of both force and rhythm is always demonstrable. In the third stage of the disease not every ventricular systole is sufficiently powerful to produce a radial pulse.

Percussion.—The transverse dullness of the heart is increased toward the left and downward as a consequence of the bilateral ventricular hypertrophy which attends the disease. So long as hypertrophy is limited to the left side of the heart, the outline of cardiac dullness at the apex remains pointed; but in the second stage of the disease, with the establishment of right ventricular hypertrophy, there is a progressive rounding of the cardiac outline at the apical area, while the dullness extends farther to the right of the sternum. In the late period of the disease, with the establishment of tricuspid insufficiency, the examiner may frequently detect an extension of cardiac dullness upward in the third and fourth intercostal spaces upon either side of the sternum, as a result of auricular dilatation.

The pulmonary bases should be carefully and methodically percussed during every examination of a subject of mitral regurgitation with the object of detecting a complicating pulmonary edema or hydrothorax. This examination commonly proves negative during the period of maintained compensation during the first and second stages of mitral regurgitation. But with the rupture of compensation in the third stage of mitral insufficiency percussion of the pulmonary bases yields impairment of vesicular resonance due to pulmonary edema, or flatness over the inferior portion of the thorax as a result of hydrothorax.

Similarly, a routine examination in this disease should not be considered complete without the careful delimitation of the areas of hepatic and splenic dullness. With the maintenance of compensation in the first and second stages of the disease these areas of dullness retain their normal dimensions; but with failure of compensation and the general venous stasis which attends this state the areas of hepatic and splenic dullness are increased in extent as a result of passive congestion of the venous radicles of these organs.

Auscultation.—Mitral regurgitation in the first period of its evolution is attended by a systolic murmur with its point of maximum intensity at the mitral area, over the apex of the heart. From this site the murmur is propagated in all directions; but it is transmitted with selective intensity toward the left axillary region; and occasionally it is audible upon the posterior aspect of the thorax near the angle of the scapula. The murmur is blowing rather than harsh and unmusical, and it partially or completely obscures the first sound of the heart at the apex. The quality and intensity of the murmur vary with the condition of the myocardium and with the posture assumed by the patient. A murmur which is faintly audible in the erect posture frequently becomes accentuated when the subject assumes the recumbent posture. During the first stage of mitral regurgitation there is no perceptible accentuation of the second sound of the heart at the pulmonic area, a negative finding which, by excluding the existence of right ventricular hypertrophy, indicates that the myocardial changes are as yet limited to the left heart.

During the second stage of the disease, on the contrary, accentuation of the pulmonic second sound is clearly perceptible, affording a perfectly reliable sign of compensation of the mitral lesion by right ventricular hypertrophy. But before basing deductions as to the extent of the mitral lesion upon the degree of accentuation of the pulmonic sound, the examiner must needs exclude the presence of obstructive disease of the lungs, and must take into consideration the age of the patient, bearing in mind the physiologic accentuation of this sound in the young subject.

During the first and second stages of the disease auscultation of the pulmonary bases yields no signs of congestion of the lungs or pulmonary edema; and so long as the mitral leakage is adequately compensated by right ventricular hypertrophy, the intensity of the mitral murmur remains uniform in intensity, in striking contrast to the enfeeblement of the murmur which is encountered in the last stage of the disease.

With the failure of compensation by the right ventricle there is a progressive enfeeblement of the accentuated pulmonary second sound, with the coincident generation of the systolic murmur of a "safety-valve leak" at the tricuspid orifice. As the systolic murmur of tricuspid insufficiency with its point of maximum intensity over the lower sternal region gains in intensity, there is a progressive enfeeblement of the mitral systolic murmur, which is audible over an ever-diminishing area, to finally become masked entirely by the increasing intensity of the tricuspid murmur.

In this late stage of the disease auscultation of the pulmonary bases yields numerous moist râles, which are universally distributed over both lungs.

Diagnosis.—The presence of a systolic murmur at the cardiac apex, which is blowing and not unmusical in quality, and is transmitted toward the left axilla and is associated wit centuation of the pulmonic second sound constitutes a clea ture of mitral regurgitation. In the early stage of the dis however, when the myocardial changes are limited to the left of the heart, accentuation of the pulmonic sound is not percep Under such circumstances the examiner should seek for disp ment of the cardiac impulse toward the left and downward curring from left ventricular hypertrophy.

In relative mitral regurgitation the examiner will usually signs of general hypertension and a murmur at the aortic or which is altogether more intense than is the mitral murmur.

The intensity of a mitral murmur to a certain degree is ind tive of the nature of the causative lesion. Thus the mi systolic murmur of acute endocarditis is soft and blowing quality and is attended by little ventricular hypertrophy; whe mitral regurgitation occurring as a result of chronic e carditis with shrinking and sclerosis of the valve segments yi a loud blowing murmur, which is attended by considerable largement of the transverse dullness of the heart.

Differential Diagnosis.—*Functional murmurs* are prone to velop at the mitral valve, although they are more frequently countered at the pulmonic valve. Nevertheless, the exami should bear in mind that the mitral valve comes second in p of frequency for these murmurs. But, as previously noted, th murmurs do not produce ventricular hypertrophy neither are t propagated beyond the limits of the precordia, and usually t are encountered in the anemic and debilitated subject.

The systolic murmur of *aortic stenosis* is frequently audible the mitral area as well as at the aortic area; but in this instance examiner will experience little difficulty in determining that point of maximum intensity of the murmur is localized in the ao area, and that its line of propagation is upward into the root the neck and not at all toward the left axillary region. Moreo the quality of the two murmurs is quite dissimilar. The mur of aortic stenosis is loud and harsh as compared with the sof "whiff" of the mitral regurgitant murmur, and aortic stenosis almost constantly attended by a palpable thrill in the aortic va area.

Pericardial friction is frequently audible over the region of cardiac apex, producing in this locality a sound with a quality unlike that of the mitral regurgitant murmur. But while p cardial friction is frequently almost synchronous with the first sound of the heart, the sound is more diffuse in its distribution, and it is never transmitted from the limits of the precordia. Neither does the sound completely replace the first sound of the heart; and moreover, it is frequently transient and evanescent and is influenced by pressure with the stethoscope and by changes in posture upon the part of the patient.

A *cardiorespiratory murmur* generated near the cardiac apex may readily be mistaken for a mitral systolic murmur during a casual examination; but this murmur does not replace the first sound of the heart; and it is markedly influenced by the respiratory excursion of the lung, being intensified during full inspiration, and frequently abolished during forced expiration.

MITRAL STENOSIS

Clinical Pathology.—Stenosis of the mitral valve is a disease of adolescence and early manhood as a rule, developing usually as a sequence of acute endocarditis of rheumatic origin. Aside from the rheumatic cases may be grouped endocarditic cases arising as a sequence of scarlatina, chorea, chlorosis, and the acute infectious fevers. But there is another group of cases of mitral stenosis in which a slow sclerosis of the valvular cusps occurs in elderly subjects of general arteriosclerosis and chronic interstitial nephritis.

The mechanical influences of the lesion in mitral stenosis are manifested in much the same manner as are those which accompany a regurgitant lesion at this valve, with certain variations which are due to the manner of inception of the primary alterations in the myocardium. As these influences operate slowly upon the myocardium and remote organs of the body, the disease may be divided into three stages, as in the case of mitral regurgitation, three stages in which one results inevitably in another when once an organic stenosis is established at the mitral orifice.

During the first stage the brunt of the lesion falls upon the left auricle and left ventricle, and during this stage of the disease the myocardial changes are confined to the left side of the heart. At the completion of ventricular systole the ventricular walls relax and during the early portion of ventricular diastole a portion of the blood which has accumulated in the left auricle during ventricular systole is aspirated into the chamber of the left ventricle. During the midportion of ventricular diastole the pressure of the blood column contained in the left auricle and the pulmonary veins forces a further quantity of the contents of the left auricle into the chamber of the left ventricle. Finally, during late ventricular diastole the systole of the left auricle propels the remainder of the blood which occupies the cavity of the auricle into the ventricle completing the first act of the cardiac cycle. In the presence of a narrowing of the mitral orifice an additional burden is thrown upon the left auricle, which hypertrophies in response to the increased demand for work. So long as the hypertrophied auricle is capable of completely emptying its contents promptly without retardation of the pulmonary circulation, the circulatory balance is adequately maintained; but as the reserve power of the auricle is limited, hypertrophy eventually gives way to dilatation, an increased burden is thrown upon the pulmonary circulation and the right heart and the second stage of the disease is ushered in with hypertrophy of the right ventricle. During this stage of the disease moderate left ventricular hypertrophy occurs as the result of the aspirating action of this chamber in drawing its proper quota of blood through the narrowed orifice, and because it must contract powerfully in order to expel the diminished output of the auricle into the aorta.

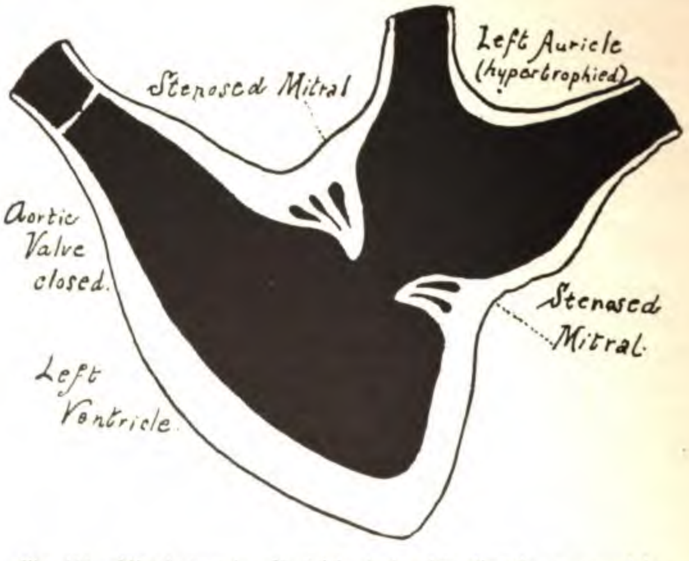


Fig. 185.—Mitral stenosis. Ventricle during late diastole or presystole.

The third stage of the disease develops when the right ventricle yields to the progressive increase in tension of the pulmonary circuit, the mechanism differing in no wise from that of mitral regurgitation.

The valvular changes in mitral stenosis present wide variations. In early endocarditic cases there are merely small vegetations upon the auricular aspects of the cusps, which produce practically no stenosis, but which are sufficient to induce "fluid veins" with the generation of a presystolic murmur. In more advanced cases the cusps are sclerosed and shrunken, frequently with adhesion of the free borders and shortening of the papillary muscles, with the reduction of the valvular orifice into a mere slit or chink, the "button-hole orifice" of Corrigan. In yet other instances the cusps are normal, but the mitral ring is reduced in caliber either as a congenital defect or as a result of secondary sclerosis.

During the first and second stages of mitral stenosis the subject of the disease experiences little discomfort and presents few symptoms. Foremost among these is moderate dyspnea which is brought out upon severe exertion. Occasionally the first intimation of the existence of the disease consists in the onset of a local paralysis from embolism of the brain. In recurrent endocarditic cases the subject is likely to present recurrent febrile attacks which develop without assignable cause. During the later stage of the disease dyspnea becomes extreme, amounting frequently to orthopnea, with free expectoration of frothy sputum which is frequently tinged with blood.

Physical Signs.—*Inspection.*—In the first stage of mitral stenosis inspection frequently proves negative, as the principal findings upon inspection in this disease depend upon embarrassment of the pulmonary circulation as a result of the mitral obstruction, and as this obstruction is adequately compensated during the early period of the disease by left auricular hypertrophy. The cardiac impulse usually occupies its normal position in the fifth left interspace; and when it is displaced by left ventricular hypertrophy at this time the displacement occurs toward the left and not downward. The area of the impulse is very frequently reduced in extent. In the case of young subjects and in women there is frequently a visible presystolic pulsation in the second and third intercostal spaces adjacent to the left sternal border, which is produced by left auricular hypertrophy.

During the second stage of the disease, with the establishment of right ventricular hypertrophy, systolic pulsation of the epigastrium is frequently to be noted, as well as a wide systolic pulsation in the third and fourth intercostal spaces along the left sternal border, developing as a result of the impact against the thoracic wall of the conus arteriosus of the right ventricle. In children and in thin chested subjects a variable degree of bulging of the precordia and lower sternal region is frequently encountered.

With failure of the right ventricle in the third stage of the disease the cardiac impulse becomes broad and undulatory along the left sternal border, together with distention of the cervical veins and frequently a positive jugular pulsation which is due to the establishment of tricuspid regurgitation. The respiratory movements of the thorax are labored, and the lips and mucous membranes exhibit cyanosis together with general pallor of the integument of the trunk and extremities. The abdomen protrudes from the accumulation of fluid in the peritoneal cavity, and the feet and ankles swell from edema of the cellular tissues.

Palpation.—Palpation of the mitral area in mitral stenosis reveals the presence of a presystolic thrill, which is pathognomic of the disease. The apex beat is of normal or slightly increased force. The thrill at the mitral area is attended by accentuation of the valve shock at the pulmonic area, which attains its maximum intensity during hypertrophy of the right ventricle during the second stage of the disease. During the third stage of the disease the mitral thrill becomes progressively enfeebled while the cardiac impulse becomes more extensive, undulatory and slapping.

The pulse of mitral stenosis is of small volume, but of high tension during the maintenance of compensation, and is disordered in both force and rhythm with the failure of the right heart. In certain cases arrhythmia is a striking feature throughout the course of the disease.

Percussion.—During the first stage of mitral stenosis percussion reveals no alteration in the cardiac outline, save in selected cases an extension of dullness in the third and fourth left interspaces arising from left auricular hypertrophy. During the second stage of the disease the area of cardiac dullness is extended toward the right, encroaching for a variable distance upon the vesicular resonance of Ebstein's cardiohepatic angle. During the third stage of the disease percussion of the pulmonary bases frequently reveals dullness from pulmonary edema; and the areas of hepatic and splenic dullness are extended as a result of chronic passive congestion of these organs.

Auscultation.—During the first stage of mitral stenosis auscultation at the apex of the heart in the vast majority of cases reveals the presence of a presystolic murmur, which is not transmitted beyond the precordia. In cases of minor stenosis, however, in which a few vegetations exist upon the auricular aspect of the valve segments, there may be no appreciable murmur, but merely a roughening and accentuation of the first sound of the heart. In these cases active exertion upon the part of the subject will frequently bring a presystolic murmur to the fore, which again becomes inappreciable when the patient has reposed for some hours in the recumbent posture.

The quality of the murmur of mitral stenosis defies adequate description; it must be heard to be appreciated. The murmur is harsh, ingravescent or crescendo in quality, increasing steadily in intensity from commencement to termination, and is followed by a sharp first sound of the heart. During the first stage of the disease, during the maintenance of left auricular hypertrophy, the murmur is generated immediately prior to ventricular systole, and the first sound of the heart at the apex, in the absence of coincident mitral regurgitation, remains clearly perceptible. The murmur of mitral stenosis is not transmitted beyond the limits of the precordia.

During the second stage of the disease, with the establishment of right ventricular hypertrophy, the mitral murmur develops slightly earlier in diastole and is well sustained throughout auricular systole, with an intensity which equals or exceeds that of the murmur during the first period of the disease. Auscultation of the pulmonic area during this stage of the disease reveals a striking accentuation of the pulmonic second sound, and not infrequently there is a reduplication of the second sound of the heart at the base.

With the failure of compensation by the right ventricle during the third stage of the disease the mitral murmur becomes progressively more feeble, to finally become inaudible or to become obscured by a systolic murmur at the tricuspid area arising as a result of tricuspid regurgitation. There is at the same time a progressive enfeeblement of the formerly accentuated second sound of the heart at the pulmonary area. Auscultation of the pulmonary bases at this stage of the disease yields numerous moist râles, which are universally distributed over both lungs.

Diagnosis.—The diagnosis of mitral stenosis is based upon the characteristic physical findings; namely, a presystolic murmur at the mitral area, ingravescent or crescendo in quality, followed by a distinct and snapping first sound of the heart, and not transmitted beyond the precordia, with increase in the transverse dullness of the heart, particularly toward the right, and with accentuation of the pulmonary second sound at the base of the heart. There are, however, a number of possible confusing findings, and a differential diagnosis is not always unattended by considerable difficulty.

Differential Diagnosis.—Foremost among the confusing factors in the diagnosis of mitral stenosis stands the ***Flint murmur*** of aortic regurgitation. In the exclusion of this possible source of error the examiner should search for the aortic organic murmur occurring at the base of the heart in this disease, the capillary pulse of Quincke and the femoral murmur of Duroziez. The quality of the two murmurs is quite dissimilar, as the Flint murmur does not possess the ingravescent and crescendo quality of the murmur of true organic mitral stenosis. While both murmurs occur at the mitral area and are not transmitted thence, the Flint murmur never possesses the same degree of intensity as is attained by the murmur of mitral stenosis.

Mitral regurgitation is attended by a soft, blowing murmur at the mitral area, which is systolic in time, and which is transmitted toward the left axilla. The progress of development of the two murmurs is, moreover, diametrically opposed, the murmur of mitral stenosis progressively increasing in intensity to terminate in an abrupt first sound of the heart, whereas the murmur of mitral regurgitation, becomes progressively less intense from its commencement with ventricular systole until its termination. Quite frequently the two murmurs are combined, the systolic murmur of mitral regurgitation ensuing shortly upon the presystolic murmur of mitral stenosis, in the presence of combined stenosis and regurgitation at the mitral orifice.

Tricuspid regurgitation produces a systolic murmur with its point of maximum intensity in the tricuspid area over the lower portion of the sternum, whence the murmur is propagated toward the right. This murmur frequently is audible in combination with the murmur of mitral stenosis late in the course of the disease; but it is attended by such striking disorders of the venous circulation that a differentiation of the two murmurs is not attended by great difficulty.

Tricuspid stenosis produces a presystolic murmur with its point of maximum intensity not far removed from that of the murmur of mitral stenosis. The quality of the two murmurs is not at all dissimilar, but stenosis of the tricuspid orifice is a rare lesion and usually forms a part of the syndrome of congenital cardiac disease.

The murmur of *aortic regurgitation* occasionally has its point of maximum intensity at the mitral area; but in this instance the time of the murmur as well as the quality serves as a basis of differentiation. The murmur of aortic regurgitation has its inception at the commencement of ventricular diastole, progressively becoming less intense during the diastolic period to become lost before the beginning of the first sound of the heart. In those cases of mitral stenosis, however, in which the mitral presystolic murmur occupies a considerable portion of the diastolic period, an error in diagnosis is very prone to be made.

PULMONARY REGURGITATION (PULMONARY INSUFFICIENCY; PULMONARY INCOMPETENCE)

Clinical Pathology.—Pulmonary regurgitation occurring as the result of organic disease of the pulmonary valve is the rarest of all acquired lesions of the heart. Isolated cases are encountered, however, in which as the result of ulcerative endocarditis attacking the right side of the heart, deformity of the valvular cusps has occurred with resulting inability of the deformed cusps to adequately close the pulmonary orifice. These cases are most frequently encountered among young adult subjects, and pulmonary insufficiency is usually combined with pulmonary stenosis. Incompetence of the pulmonary valve is one of the rare congenital defects of the heart.

Relative pulmonary regurgitation, on the contrary, in which regurgitation occurs at this orifice in the absence of structural derangement of the valve, is of frequent occurrence. Relative pulmonary insufficiency may be of intracardiac or extracardiac origin. In the presence of uncompensated lesions of the left side of the heart, with the progressive rise in tension in the pulmonary circulation, the right ventricle is prone to yield with resulting enlargement of the ring supporting the cusps of the semilunar valve and the induction of relative pulmonary insufficiency. Similarly, in the presence of obstructive disease of the lungs of prolonged duration, such as chronic ulcerative phthisis, cirrhosis of the lung, and hypertrophic emphysema, the persistent hypertension in the pulmonary circulation is capable of inducing relative insufficiency of the pulmonary valve.

The immediate effect of regurgitation at the pulmonary valve is to induce transient dilatation of the right ventricle, which is compensated by hypertrophy of its muscular walls. So long as compensation is maintained the circulatory imbalance is corrected; but the ventricle eventually reaches the limit of its reserve power, whereupon dilatation beyond the power of compensation follows, with the establishment of tricuspid regurgitation, and general engorgement of the venous system. As a result of stasis in the pulmonary circulation edema of the lungs ensues and the subject exhibits the signs of chronic bronchial catarrh.

Physical Signs.—*Inspection.*—In its early stages simple pulmonary regurgitation yields no evidence of its existence upon inspection. As the disease progresses the cardiac impulse is displaced toward the left axillary line but is not displaced downward. While right ventricular hypertrophy is maintained, there is visible pulsation of the epigastrium, and in thin chested individuals there is visible bulging of the lower portion of the precordia. As the disease progresses the subject becomes dyspneic with chronic cough and serous expectoration, while the trunk and extremities are pallid as a result of deficient arterial circulation. With the establishment of tricuspid insufficiency the positive jugular pulse becomes demonstrable in the jugular veins.

Palpation.—During the early period of the disease with the maintenance of right ventricular hypertrophy the apex beat is diffuse and extends downward toward the epigastrium and well inward toward the left sternal border. A forceful, lifting systolic pulsation is usually to be elicited in the upper epigastrium as a result of the powerful contractions of the right ventricle. Similarly, the right costal arch is occasionally raised by the impact of the overacting right ventricle against the liver. Late in the disease a true expansile pulsation of the liver can occasionally be elicited upon bimanual palpation of the liver, occurring as a manifestation of tricuspid regurgitation. Occasionally it is possible to elicit a fine diastolic thrill at the pulmonary area; but the thrill is not constantly present, neither is it detected with the same constancy as is the case of the systolic thrill accompanying stenosis of this valve.

The *pulse* in pulmonary regurgitation is apt to be of low tension and diminished volume and arrhythmic, but presents nothing which is characteristic of the disease.

Percussion.—The transverse dullness of the heart is increased toward the right in direct proportion to the degree of hypertrophy of the right ventricle. As dilatation ensues upon compensatory hypertrophy the right margin extends well to the right of the sternum. Late in the disease the pulmonary resonance is impaired over the bases of the lungs, and the areas of dullness of the liver and spleen are more extensive than in the normal subject.

Auscultation.—Auscultation of the pulmonary area reveals the presence of a blowing murmur with its point of maximum intensity at the junction of the second left costal cartilage with the sternum, whence it is transmitted downward along the left sternal border and toward the cardiac apex. The murmur is commonly blowing and musical, but in cases of associated pulmonary stenosis and regurgitation the murmur is apt to have a harsher quality and to completely replace the second sound of the heart at the pulmonary area. In the presence of relative pulmonary insufficiency auscultation of other valve areas will elicit the murmurs peculiar to the organic lesions which are present.

Diagnosis.—The diagnosis rests upon the detection of a diastolic murmur with its point of maximum intensity at the pulmonary area, whence it is transmitted downward, with signs of hypertrophy or dilatation of the right heart, and general venous stasis. In reaching a diagnosis of simple pulmonary regurgitation the examiner should bear in mind the relative infrequency with which this valve is attacked by organic disease.

PULMONARY STENOSIS

Clinical Pathology.—Stenosis at the pulmonary valve is in the vast majority of cases due to a congenital defect and is associated with other congenital deformities of the heart such as tricuspid stenosis, patent ductus arteriosus, patent foramen ovale, or perforation of the ventricular septum. Very rarely the lesion is the sequence of ulcerative endocarditis attacking the pulmonary valve.

But while true stenosis of the pulmonary valve is a rare lesion, there are many pathologic conditions capable of inducing the generation of a systolic murmur at this valve. First and foremost among these factors stands anemic states with the induction of functional murmurs, which are more frequently encountered at the pulmonary valve than at the other valves of the heart. Similarly, dilatation of the pulmonary artery distal to the valve produces a condition of *relative pulmonary stenosis,* which is attended by a systolic murmur at this area in the absence of organic disease of the valve. Compression or constriction of the pulmonary artery by new growths, enlarged glands, or pleural adhesions are all capable of inducing a murmur which may be mistaken for that of pulmonary stenosis. The nervous imbalance of the cardiac mechanism incident to exophthalmic goiter frequently is betrayed by a systolic murmur at the pulmonary valve; and shifting of the mediastinal structures through cardiac displacement may produce torsion of the vessel with a resulting systolic murmur in the vicinity of the pulmonary orifice.

The immediate effects of the lesion in stenosis at the pulmonary valve are expended upon the right ventricle, which hypertrophies to compensate for the obstruction; and if the obstruction is slight, no further myocardial change may be induced. In the presence of a greater degree of stenosis however, the persistent increase of intraventricular pressure results in right ventricular dilatation, relative tricuspid insufficiency and general venous engorgement.

Physical Signs.—*Inspection.*—In cases of pulmonary stenosis of congenital origin, the signs upon inspection are largely those of congenital heart disease, notably extreme cyanosis, dyspnea and and clubbing of the fingers. In the presence of minor degrees of stenosis, dyspnea and a little cyanosis upon active exertion may remain the sole signs of the disease. When right ventricular hypertrophy is a marked feature of the case the cardiac impulse is displaced toward the left and is visible also farther toward the right as a systolic pulsation of the upper portion of the epigastrium. With the supervention of tricuspid insufficiency the positive venous pulse becomes appreciable in the jugular veins.

Palpation.—Palpation of the pulmonary valve area reveals the presence of a fine systolic thrill, which is frequently diffused over a wider area than is the case with the systolic thrill of aortic stenosis. The thrust of the right ventricle is clearly palpable in the upper epigastrium in the presence of hypertrophy of this chamber of the heart, and its impact not infrequently communicates a systolic impulse to the adjacent liver.

The *pulse* at no time during the course of the disease presents any changes which are characteristic of the disease. With failure of the right heart its volume is reduced and its rhythm is disturbed, findings which are in no wise distinctive of the lesion present.

Percussion.—Delimitation of the cardiac outline proves negative during the early stages of pulmonary stenosis, and in the presence of minor degrees of stenosis which are readily compensated by a slight degree of right ventricular hypertrophy the outline is little altered throughout the disease. With the supervention of frank ventricular hypertrophy, the outline is increased toward the right; and in the presence of right ventricular and auricular dilatation the increase toward the right is further accentuated.

With the establishment of tricuspid regurgitation and venous stasis the dullness of the liver and of the spleen is appreciably increased in extent.

Auscultation.—Pulmonary stenosis is attended by a systolic murmur with its point of maximum intensity at the pulmonary area, whence it is diffused in all directions, but with selective intensity upward toward the root of the neck, but it does not extend into the vessels of the cervical region as is the case with the systolic murmur of aortic stenosis. The murmur is audible over a considerable area of the precordia, but with care its point of maximum intensity can be localized to the second left intercostal space adjacent to the sternum.

The quality and intensity of the murmur vary with the degree of stenosis and with the power of the ventricular contraction. The murmur is inherently harsh and unmusical, in marked contrast to the soft and blowing murmurs of functional origin occurring at this valve. In the presence of minor grades of stenosis, however, the intensity of the murmur is not great and its duration is not considerable. In the case of marked stenosis, on the contrary, the murmur is harsh, intense, and sustained, commencing with ventricular systole and terminating with the second sound of the heart, which is not infrequently replaced by the diastolic murmur of a coexistent pulmonary regurgitation.

With the establishment of tricuspid insufficiency, the systolic murmur of this lesion is perceptible at the tricuspid area, obscuring in this locality the first sound of the heart.

Diagnosis.—The diagnosis of simple pulmonary stenosis rests upon the cardinal signs of the disease; namely, a systolic murmur with its point of maximum intensity at the pulmonary area, whence it is transmitted upward, but which does not extend into the great vessels of the neck; a palpable thrill at the same area, which is to be detected in the vast majority of cases; and signs of right ventricular hypertrophy or of right heart failure.

Differential Diagnosis.—*Functional murmurs,* which are encountered at the pulmonary valve more frequently than at the other valve areas of the heart, are systolic in time; but they are soft and blowing; they are transient, coming and going at successive examinations; they are not transmitted beyond the limits of the precordia; and they do not induce changes in the myocardium or the character of the pulse.

In the *spurious murmurs* which are generated at this area by torsion or dilatation of the pulmonary artery, by the constriction of the vessel by pleural adhesions, or by the disturbance of exophthalmic goiter, there is no evidence of circulatory disturbance or of myocardial changes. The patient is not dyspneic and is apparently in a good state of health. The murmurs, moreover, are more diffuse than is the murmur of simple pulmonary stenosis, and they exhibit no selective line of transmission.

Aneurysm of the aorta produces a systolic murmur which is audible in the vicinity of the pulmonary area; but the murmur and the thrill which accompanies it has a more diffuse distribution upon the anterior thoracic surface; it is commonly attended by local pulsation and by dullness over its distribution, and its systolic murmur is transmitted into the vessels of the neck, which is not true of the murmur of pulmonary stenosis.

Patency of the ductus arteriosus induces a murmur in the vicinity of the pulmonary area; but the point of maximum intensity of this murmur is farther from the sternal margin than is the case with the pulmonary stenotic murmur; and, moreover, the murmur of a patent ductus arteriosus continues beyond the second sound of the heart, which is not characteristic of the murmur of pulmonic stenosis.

TRICUSPID REGURGITATION (TRICUSPID INSUFFICIENCY; TRICUSPID INCOMPETENCE)

Clinical Pathology.—Regurgitation occurs at the tricuspid orifice in two distinct forms; namely, as simple organic insufficiency of the valve, and as relative or functional insufficiency of the tricuspid valve. Organic disease of this valve of the heart is indeed rare, as the predilection of endocarditic changes is for the left side of the heart. Occasionally tricuspid regurgitation arises from congenital malformation of the valve, in which event it is usually combined with tricuspid stenosis.

If organic insufficiency at the tricuspid valve is infrequent, relative regurgitation at this orifice, arising as a result of right ventricular dilatation under the stress of heightened tension in the pulmonary circulation, is a very frequent disease. Under these circumstances the tricuspid valve develops a "safety-valve leak" with the generation of a soft and blowing systolic murmur with its point of maximum intensity at the tricuspid area over the lower end of the gladiolus, the valvular disease constituting merely a link in the syndrome of chronic valvular disease of the heart. Indeed, even in the absence of incompetence upon the part of the valves of the left heart, tricuspid regurgitation may be induced by persistent hypertension in the pulmonary circulation, which arises as a result of obstructive disease of the lungs during the course of chronic ulcerative phthisis, chronic interstitial pneumonia, or hypertrophic emphysema.

The primary effect of the tricuspid lesion is an immense dilatation of the right auricle, the reserve power of which is so limited that auricular hypertrophy never adequately compensates for the lesion at the tricuspid valve. Venous engorgement appears early in the disease, with overfullness of the cervical veins, systolic pulsation of the liver, the positive venous pulse in the jugulars, anasarca and general edema of the extremities.

Physical Signs.—*Inspection.*—In tricuspid regurgitation the physical signs vary with the state of the myocardium and with the syndrome of which tricuspid regurgitation is a part. In the presence of simple isolated tricuspid regurgitation of congenital or endocarditic origin the cardiac impulse is carried toward the right as a result of right ventricular hypertrophy, frequently occupying a position behind the sternum. In relative tricuspid insufficiency occurring as part and parcel of left-sided valvular disease, on the contrary, the cardiac impulse is displaced to the left and downward, at the same time encroaching upon the upper epigastrium, in which site it is heaving and forceful if right ventricular hypertrophy is maintained. As the disease progresses, dilatation of the right auricle produces a visible pulsation along the right sternal border in the second, third, and fourth interspaces.

Whatever may be the cause of the lesion, tricuspid insufficiency produces multiple signs of venous engorgement and general venous stasis. The subject is dyspneic and frequently cyanotic about the face, with anemic pallor of the trunk from deficient arterial circulation. The positive venous pulse is demonstrable in the jugular veins, and the abdomen is prominent from ascites, while the feet and ankles swell from edema.

Palpation.—Palpation of the upper epigastrium reveals a systolic pulsation, arising as the result of the impact of the right ventricle. In simple organic regurgitation of the tricuspid valve this pulsation does not possess the same degree of force which is exhibited in the secondary or relative form of the disease, in which right ventricular hypertrophy is more extreme. Bimanual palpation of the liver reveals the presence of a true, expansile pulsation of this organ which is synchronous with ventricular systole. A pseudopulsation of the liver is sometimes caused by the impact of the hypertrophied right ventricle against the liver, and should not be mistaken for the true expansile pulsation of the organ, which is a very good sign of tricuspid regurgitation.

The *pulse* of tricuspid regurgitation presents no distinctive characteristics. Asymmetry of the radial pulses is apt to arise as the result of compression of the right subclavian artery by the enlarged right auricle.

Percussion.—In simple tricuspid regurgitation, the cardiac outline extends well to the right of the sternum, with little or no enlargement of the left ventricle. In relative tricuspid regurgitation, on the contrary the transverse dullness of the heart is extended both toward the right and toward the left and downward toward the upper epigastrium as a result of combined ventricular hypertrophy or dilatation.

Percussion of the base of the thorax reveals impairment of the vesicular resonance as a result of pulmonary edema or frank dullness late in the disease from hydrothorax. The areas of hepatic and splenic dullness are extended in the late stages of the disease.

Auscultation.—Tricuspid regurgitation is attended by a blowing systolic murmur with its point of maximum intensity at the tricuspid area over the lower portion of the gladiolus, whence it is transmitted toward the right and slightly upward. The point of maximum intensity of this murmur is not as discrete and circumscribed as is the case with endocardial murmurs occurring at the other valves of the heart. Frequently the blowing murmur is audible over the greater portion of the precordia; but careful examination will usually serve to localize its maximum intensity over the lower sternal region and to reveal a selective transmission of the murmur toward the right and upward. The first sound of the heart is obscured by the murmur, but is seldom entirely replaced by it. The second sound varies with the concomitant state of the myocardium. In simple isolated tricuspid regurgitation the second sound is unaltered in intensity or slightly enfeebled, whereas in relative tricuspid insufficiency the second sound is accentuated during the maintenance of right ventricular hypertrophy, to become enfeebled with the supervention of right ventricular dilatation.

Diagnosis.—The cardinal signs of tricuspid regurgitation are a systolic murmur with its point of maximum intensity at the tricuspid area, whence it is transmitted toward the right and slightly upward, signs of right ventricular hypertrophy, the positive venous pulse in the jugulars, the systolic hepatic pulse, and signs of general venous engorgement. In cases of relative tricuspid regurgitation the murmurs of the causative left-sided valvular lesions are to be detected.

TRICUSPID STENOSIS

Clinical Pathology.—Stenosis at the tricuspid orifice ranks among the rarest of the valvular lesions of the heart. The majority of the cases have occurred in female subjects, and the lesion is rarely diagnosed during life.

Most cases of tricuspid stenosis are of congenital origin, the segments by fusion of their free borders leaving a mere buttonhole or chink at the tricuspid orifice. There are cases, however, which are of endocarditic origin, and which are associated with similar stenotic lesions at the mitral orifice.

The primary effect of the stenotic lesion is to add to the burden of the right auricle, which hypertrophies in response to the increased demand for work. In cases of moderate stenosis of the valve this compensatory hypertrophy is adequate and is maintained and there is no embarrassment of the venous circulation. In more pronounced cases, however, right auricular dilatation eventually supervenes, with signs of general venous stasis, and effusion into the serous sacs and the cellular tissues of the body.

Physical Signs.—*Inspection.*—Many cases of tricuspid stenosis with adequate compensation yield no physical signs for years or those which are exhibited are obscure and conflicting. Signs which point to stenosis of the tricuspid orifice comprise persistent moderate cyanosis, with distention and tortuosity of the cervical veins, and occasionally a pronounced negative venous pulse in the jugular veins.

Palpation.—Tricuspid stenosis is occasionally attended by a presystolic thrill which is palpable at the tricuspid area over the lower end of the sternum, but the thrill is not a constant finding in this disease. The cardiac impulse is usually encountered in its normal site, and its force and extent are not deranged in many cases. Pronounced stenosis of the valve, however, causes diminution in the force and extent of the apex-beat, and its rhythm is apt to be deranged.

The *pulse* is in no wise characteristic of the disease. In the presence of pronounced stenosis its volume is reduced and it is apt to be accelerated and disordered in rhythm.

Percussion.—The cardiac outline is unaltered in many cases, while in the presence of right auricular dilatation the transverse dullness of the heart is extended to the right of the sternum in the third and fourth interspaces.

Auscultation.—The lesion of tricuspid stenosis generates a presystolic murmur with its point of maximum intensity at the tricuspid area over the lower end of the gladiolus, whence it is not transmitted beyond the limits of the lower precordia. In the presence of marked stenosis of the valve the second sounds of the heart are diminished in intensity at the aortic and pulmonary areas at the base of the heart.

Diagnosis.—The detection of a presystolic murmur with its point of maximum intensity in the tricuspid area and frequently a presystolic thrill in the same region, with signs of venous engorgement point to tricuspid stenosis. As tricuspid and mitral stenosis are frequently concomitant lesions, the mitral murmur frequently obscures the tricuspid murmur and leads to an error in diagnosis

CHAPTER XX

DISEASES OF THE MYOCARDIUM

ACUTE MYOCARDITIS (ACUTE MYOCARDIAL DEGENERATION)

Clinical Pathology.—Acute myocarditis occurs clinically in two forms; namely, as *acute parenchymatous myocarditis;* and as *acute interstitial myocarditis.*

Acute parenchymatous myocarditis occurs as a complication of acute infectious febrile diseases, notably pneumonia, typhus and typhoid fevers, scarlatina, and diphtheria. An acute myocarditis arising during these diseases usually has its inception during the active febrile stage of the disease, at which time it may be the cause of sudden death; but in other instances its incidence is deferred, the disease developing and occasionally causing death during late convalescence. Acute parenchymatous myocarditis also occurs as a complication of endocarditis and pericarditis, by secondary infection of the myocardium from the causative organism of these diseases.

While the name myocarditis implies an inflammation, the signs of acute inflammation are not in evidence, the characteristic changes in the myocardium being those of a slow degeneration or metamorphosis, ranging in gravity from cloudy swelling in the favorable cases to fatty degeneration in the grave cases.

Grossly the heart presents a varied appearance, being pale or grayish-red in the presence of cloudy swelling; yellowish in fatty degeneration, and gray in the presence of extensive hyaline change. It is probable that the earliest retrogressive change in acute parenchymatous myocarditis, in those cases which yield the best prognosis, is cloudy swelling; and that the ultimate change is fatty degeneration, in which the prognosis is extremely grave.

Acute interstitial myocarditis also develops during the course of acute fevers, Leyden first describing the disease in connection with scarlatina. Rhomberg has noted the changes characteristic of the disease in diphtheria, typhoid fever, acute rheumatic fever, and variola. The disease occurs in two grades of severity; namely, the *transient nonsuppurative interstitial myocarditis;* and the more grave *suppurative interstitial myocarditis.* In the former the inter-muscular spaces of the myocardium are infiltrated with leucocytes, the coronary capillaries are dilated, and the muscle bundles present areas of vacuolation, nuclear multiplication, and pigmentation; but the morbid process frequently eventuates in resolution without the formation of fibrous connective tissue between the muscle bundles.

The suppurative form of the disease, of more grave prognostic import, is usually the sequence of infectious embolism of the coronary arteries. When one of the terminal vessels of this arterial system becomes occluded by a simple, noninfectious embolus, anemic or hemorrhagic infarction of the myocardium is apt to ensue; but if the occluding embolus contains infectious material, if its source happens to be a vegetation from the cusp of a cardiac valve which is the site of infective endocarditis, or from an area of infective osteomyelitis, a more serious lesion of the myocardium ensues. At the sites of obstruction circumscribed areas of leucocytic infiltration and bacterial colonization develop, leading to minute purulent infiltrations between the muscle bundles, weakening the heart wall and predisposing to aneurysm and rupture. The rupture may occur externally into the pericardial sac with the production of purulent pericarditis; or the pus may discharge into a cavity of the heart, the organisms being distributed to various organs of the body by the blood stream with the production of metastatic abscesses.

Instead of terminating by resolution as in the case of acute nonsuppurative interstitial myocarditis, the areas of purulent infiltration are prone to result in the formation of fibrous patches, weakening the cardiac musculature and predisposing to aneurysm of the cardiac wall.

Physical Signs.—Physical signs in this disease are often meager, and when present, are not clear and distinctive. The cardiac impulse as a rule shows a primary accentuation, with a subsequent weakening in its force and area. In certain cases sudden death may occur either during the height of an acute infectious disease or even late in convalescence. A fairly reliable diagnostic sign is an equalization of the intensity of the first and second sounds of the heart, both sounds assuming a valvular quality, with coincident signs of pulmonary stasis. Late in the course of the disease, when cardiac dilatation has supervened, the area of cardiac dullness is extended, and a relative mitral regurgitant murmur is frequently audible.

Diagnosis.—The diagnosis must be reached through the discovery of a causative factor rather than upon the physical signs, which are often few and confusing. But when during the course of an acute infectious disease the heart tones become enfeebled, equalized, and valvular, with alterations in rhythm as embryocardia or gallop-rhythm, the incidence of an acute myocarditis may be inferred.

Many of the cases simulate closely acute endocarditis; but in the latter disease the cardiac weakness is not so rapid and extreme, neither is the cardiac rhythm disturbed to an equal degree.

CHRONIC MYOCARDITIS (CHRONIC FIBROUS MYOCARDITIS; CHRONIC INTERSTITIAL MYOCARDITIS)

Clinical Pathology.—Chronic myocarditis is a slow sclerosis of definite areas of the myocardium, developing secondarily to changes in the coronary arteries. The essential lesion of the coronary circulation which is productive of chronic myocarditis is a narrowing or obliteration of the lumen, the result of obliterative endarteritis or embolic occlusion. Obliterative endarteritis of the coronary circulation is merely part and parcel of generalized arteriosclerosis.

When a noninfectious embolus lodges in a terminal branch of a coronary artery, an infarct of the myocardium is apt to form. The infarct is surrounded soon by a zone of infiltrating leucocytes. The infarct is wedge-shaped with the apex of the wedge directed toward the site of the embolism. The infarct, which may assume either the anemic or hemorrhagic type, leads to an area of softening of the myocardium to which the term *myomalacia cordis* has been applied by Ziegler. This area is a point of lowered resistance to the endocardiac pressure and is liable to lead to rupture with sudden death. If rupture does not occur, the area of infarction is gradually replaced by fibrous connective tissue.

The portions of the myocardium usually attacked are the lower two-thirds of the anterior wall and the upper portion of the posterior wall of the left ventricle; the interventricular septum; and the bases of the papillary muscles.

Associated changes occurring in the heart which is the seat of chronic myocarditis comprise hypertrophy, dilatation, and valvular disease. In cases of moderate involvement there is sufficient sound myocardium to compensate for the cardiac impairment by hypertrophy. In more extensive fibrosis, however, there is little intact myocardium; the cardiac wall yields under the normal or increased endocardiac pressure; and dilatation becomes inevitable. Valvular disease develops in association with moderate cases of fibrosis when it involves the papillary muscles, even in the presence of cardiac hypertrophy. In cases in which the sclerotic process is extreme and associated with cardiac dilatation, valvular disease is a constant accompaniment.

Physical Signs.—The physical findings in chronic myocarditis are not characteristic and distinctive, as they vary with the stage of the disease and with the state of the myocardium and the cardiac valves. As in the acute form of the disease, the heart sounds are usually less muscular and more valvular in quality; they are accentuated in the presence of compensatory hypertrophy; and they are enfeebled after dilatation has supervened. The aortic second sound is frequently accentuated, as the patients often have arteriosclerosis. The pulse is hard and tense, frequently with stiff, unyielding arterial wall, and is often disordered in rhythm. The area of cardiac dullness is often found extended as a consequence of cardiac hypertrophy or dilatation. Murmurs are encountered in the purely valvular cases and in cases associated with cardiac dilatation.

Diagnosis.—The diagnosis of chronic myocarditis is based partially upon the physical signs, but principally upon the discovery of arteriosclerosis in a subject past middle life who presents such physical findings. Chronic dyspnea and signs of pulmonary congestion aid in establishing the diagnosis.

CARDIAC HYPERTROPHY

Clinical Pathology.—An overgrowth of the musculature of the heart with maintenance of its nutrition may involve a single chamber of the heart, one side of the heart, or the entire organ. The portion which is most frequently involved is the left ventricle.

Thickening of the wall of the heart with enlargement of the chamber is termed eccentric hypertrophy. A similar mural change with decrease in the size of the chamber is termed concentric hypertrophy, a condition which has not been demonstrated to exist to the satisfaction of many clinicians.

The cause of cardiac hypertrophy is increased work thrown upon the heart while its nutrition is maintained. The causes of this overwork may reside within the heart, or without the viscus. The persistent and continuous muscular exertion of the athlete and of the person who is engaged in a laborious occupation is frequently provocative of cardiac hypertrophy. Habitual overeating and the ingestion of excessive quantities of beer have been followed by hypertrophy of the heart. Interference with the cardiac action by pericardial adhesions in chronic adhesive pericarditis is constantly attended by more or less hypertrophy of the heart, as are diseases of innervation of the heart which lead to continual cardiac overaction.

Arteriosclerosis and chronic nephritis by raising the blood pressure in the greater circulation lead to hypertrophy of the left ventricle; while obstructive disease of the lung such as hypertrophic emphysema, chronic interstitial pneumonia, and phthisis, by raising the pressure in the lesser circulation lead to hypertrophy of the right ventricle. Left-sided valvular lesions are productive of hypertrophy of the left ventricle and ultimately of the right ventricle. Left auricular hypertrophy is caused by regurgitant and stenotic lesions of the mitral valve; and right auricular hypertrophy follows similar lesions affecting the tricuspid valve. Pregnancy is frequently attended by moderate hypertrophy of the heart.

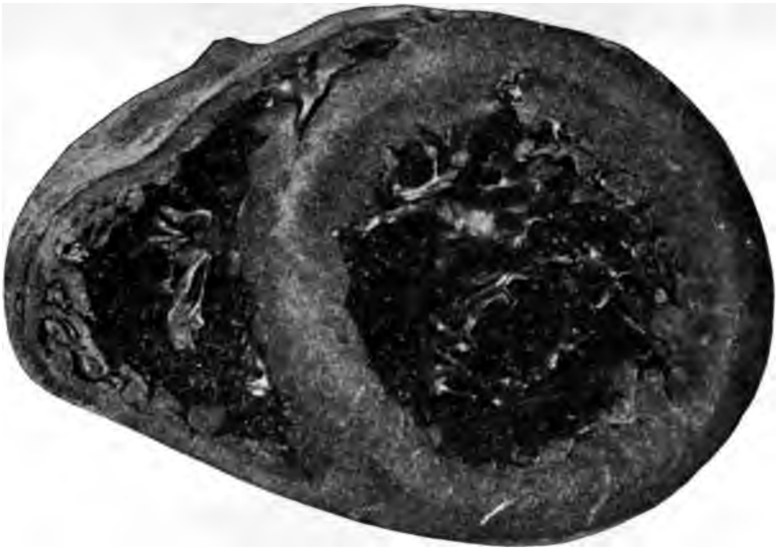


Fig. 186.—Enormous hypertrophy of left ventricle due to prolonged increased peripheral resistance. Note that the whole anterior surface of the heart is occupied by the left ventricle. The right ventricle does not appear to be much affected. (From Warfield.)

The hypertrophied heart is increased in size in one or more directions. occasionally reaching such an extent as to constitute the cor bovinum. The shape of the hypertrophied heart varies in different types of hypertrophic change. In total hypertrophy of the heart the organ is roughly round or spherical. In left ventricular hypertrophy and in right ventricular hypertrophy these portions of the heart are respectively enlarged.

LEFT VENTRICULAR HYPERTROPHY

Physical Signs.—*Inspection.*—In left ventricular hypertrophy the cardiac impulse is forcible and heaving and is displaced downward and toward the left. Precordial bulging is noticeable in children and in female subjects with thin chest walls. There is frequently visible pulsation of the carotid arteries. The area of the cardiac impulse is extended.

Palpation confirms the displacement of the cardiac impulse and its firm, heaving character. The valve shock over the aortic valve is exaggerated. The pulse is regular, of full volume, and of high tension. The cardiac impulse is not increased in frequency, but it is powerful and heaving. The impulse may be localized in the sixth or seventh interspace as far outward as the anterior axillary line. In the presence of hypertrophic emphysema it may be impossible to palpate the apex-beat owing to the intervention of the anterior pulmonary borders between the heart and the chest wall.

Percussion reveals an increase in the transverse dullness of the heart toward the left and downward, the left border of the dullness frequently extending past the anterior axillary line, and the lower limit occupying the seventh intercostal space.

Auscultation of the precordia reveals accentuation and occasionally reduplication of the aortic second sound. The first sound at the apex is similarly accentuated. There is in certain cases a systolic murmur at the mitral area due to incompetence of the mitral valve. Occasionally in left ventricular hypertrophy there is a peculiar, tinkling sound audible to the right of the cardiac apex. A cardiorespiratory murmur may be encountered in certain instances due to the strong impact of the hypertrophied ventricle against a portion of the lung which is anchored anterior to the heart by pleural adhesions. When the cardiac valves are normal the first sound of the heart, in addition to its accentuation, exhibits a perceptible prolongation owing to the slow and powerful contraction of the hypertrophied ventricle. When the hypertrophy is caused by or is associated with valvular lesions, or when dilatation is imminent, relative murmurs may be heard.

RIGHT VENTRICULAR HYPERTROPHY

Physical Signs.—*Inspection.*—In hypertrophy of the right ventricle there is undue prominence of the lower sternum and epigastrium, frequently combined with systolic epigastric pulsation. The condition is also attended by systolic pulsation along the right sternal border in the sixth and seventh interspaces. Pulsation is not infrequently noted above these levels as well, due to right auricular hypertrophy. The cardiac impulse is displaced to the right, possibly lying behind the sternum or to the right of this bone.

Palpation reveals the presence of pulsation at the lower end of the sternum and in the epigastrium; but the thrust is not as strong or as distinct as it is in the case of left ventricular hypertrophy. The valve shock over the pulmonary valve is stronger than is that over the aortic area. There is occasionally a palpable impulse transmitted to the liver by the overacting right ventricle, which should not be confused with the expansile systolic pulsation of that organ which occurs with tricuspid regurgitation.

Percussion.—Cardiac percussion shows that the area of cardiac dullness is extended toward the right, occasionally extending an inch to the right of the sternum.

Auscultation.—The tricuspid first sound is accentuated and somewhat prolonged, and a systolic "safety-valve" leak is occasionally demonstrable when right ventricular failure is imminent. The pulmonic second sound is invariably accentuated and reduplication of the second sound of the heart is frequently encountered.

The pulse is of small volume, but is regular, unless dilatation is imminent, in which event the rhythm of the pulse is disturbed.

LEFT AURICULAR HYPERTROPHY

Physical Signs.—Left auricular hypertrophy is seldom to be ascertained by physical means. An extension of cardiac dullness to the left of the sternum in the second and third interspaces is suggestive when found; and, if the hypertrophy is due to mitral stenosis, the presystolic murmur of this condition may be audible. Or, in the event that the auricular hypertrophy is secondary to mitral regurgitation, a systolic murmur may be audible over the cardiac apex.

RIGHT AURICULAR HYPERTROPHY

Physical Signs.—In hypertrophy of the right auricle physical signs are meager, but there is apt to be an increase in the area of cardiac dullness to the right of the sternum in the second, third and fourth interspaces. Presystolic pulsation along the right sternal border in this region is occasionally encountered. These signs, with the signs of right ventricular hypertrophy and with a systolic murmur at the tricuspid valve area, are very suggestive of right auricular hypertrophy. Systolic pulsation in the jugular veins, with signs of general venous engorgement point to tricuspid regurgitation with right auricular hypertrophy.

Diagnosis.—Cardiac hypertrophy is indicated by the powerful, heaving character of the cardiac impulse; the increase in the area of cardiac dullness in one or more directions; the accentuation of the second sounds of the heart; and the hard tense pulse of full volume. However, certain other intracardiac and extracardiac conditions rather closely simulate cardiac hypertrophy and require differentiation.

Fibroid retraction of the left lung may cause a wide impulse, which may during a casual examination suggest cardiac hypertrophy. But the physical signs of cirrhosis of the lung in this instance are quite sufficient to render a differential diagnosis comparatively simple.

Neurotic conditions incident to exophthalmic goiter, and the excessive ingestion of stimulants, as tea, coffee, or tobacco, cause transient vigorous cardiac action, simulating cardiac hypertrophy; but in these conditions the impulse is less diffuse and heaving; the cardiac dullness is not extended; and there are ameliorations and aggravations of the attacks.

In pericardial effusion the increased area of dullness is triangular or pear-shaped with the base directed downward toward the diaphragm; and the heart sounds are enfeebled, and the pulse is of the pulsus paradoxus type.

In cardiac dilatation the heart sounds are feeble, but distinct, and the pulse is feeble and irregular, while cardiac murmurs are to be detected at one or more of the valve areas. At the same time there are signs of general venous stasis incident to right heart failure.

CARDIAC DILATATION

Clinical Pathology.—Cardiac dilatation is an enlargement of one or more chambers of the heart, occurring as a result of yielding of the myocardium which is the seat of secondary changes. Clinically cardiac dilatation may be said to exist when a chamber of the heart is no longer able to empty itself during systole. The underlying cause of cardiac dilatation resides in an increase in intracardiac tension or in inherent degeneration of the cardiac wall; and in most instances there is a combination of these two factors operative in the induction of cardiac dilatation.

Physical exertion operates in two ways to produce cardiac dilatation; namely, in the form of prolonged muscular exertion in the form of mountain climbing or foot racing, and in the form of sudden, abrupt exertion in the presence of valvular heart disease or chronic myocarditis.

Valvular lesions of the heart are the most fertile sources of dilatation of the several chambers of the heart, the chamber passing through the usual cycle of temporary dilatation, transient compensatory hypertrophy, and permanent dilatation.

Myocarditis, induced by malnutrition in anemia or chronic obliterative endarteritis or by the chronic irritative action of the toxins of syphilis and plumbism, or by the direct effects of the toxemia of acute infections, predisposes to dilatation of the heart.

Increased peripheral resistance to the onward flow of the blood stream through the greater or lesser circulation tends to result in ultimate dilatation of the chamber of the heart which bears the brunt of the burden: arteriosclerosis and chronic nephritis raising the blood pressure in the general circulation, and obstructive disease of the lungs or left-sided valvular lesions of the heart acting similarly upon the pulmonary circulation.

Ventricular dilatation is associated with relative insufficiency caused by yielding of the fibrous ring of the auriculoventricular orifice, with the result that although the valvular cusps are not deformed, they are yet unable to close the abnormally large orifice. Moreover, the chordæ tendineæ and papillary muscles do not share in the dilatation, and remaining of normal length, they do not permit the accurate coaptation of the free borders of the valve cusps.

Dilatation may occur with thinning of the cardiac wall or in association with compensatory hypertrophy with thickening of the myocardium. Dilatation with thinning of the myocardium is an acute process, usually observed in the right ventricle when this chamber is subjected to a sudden hypertension in the pulmonary circulation. Dilatation with hypertrophy, on the contrary, is a slower and more progressive change, associated with slowly developing and maintained hypertrophy of the myocardium in response to an increased demand for work.

As in the case of cardiac hypertrophy, so in the presence of cardiac dilatation the contour of the heart varies with the type and the degree of dilatation. When dilatation involves all four chambers of the heart, the heart is roughly spherical in outline. When a single chamber or one side of the heart is dilated, the contour is correspondingly irregular. The right ventricle is anatomically liable to a greater degree of dilatation than is the left ventricle, and the right auricle to more than the left auricle.

Physical Signs.—*Left Ventricular Dilatation.*—Upon *inspection* the cardiac impulse is observed to be displaced toward the left and downward, and is diffuse and frequently undulatory in character. Not infrequently a cardiac impulse cannot be defined upon inspection in the late stage of dilatation of the left ventricle.

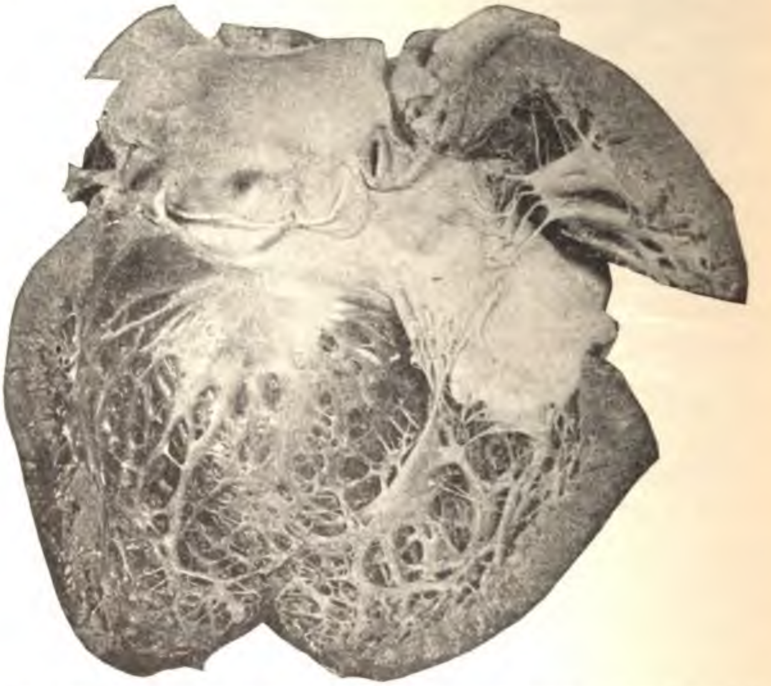


Fig. 187.—Aortic incompetence with hypertrophy and dilatation of left ventricle, the result of arteriosclerosis affecting the aortic valves. Note how the valves have been curled, thickened, and shortened, the edges of valves being a half inch below the upper points of attachment. The anterior coronary artery is shown, the lumen narrowed. (Reduced one-half.) (From Warfield.)

Upon *palpation* the cardiac impulse is defined with difficulty, it possesses little lifting power, and in late cases it may be altogether impalpable. A visible diffuse cardiac impulse which is not readily palpable is of immense diagnostic significance.

Upon *percussion* the transverse dullness of the heart is extended toward the left and downward to a degree which is commensurate with the dilatation of the left ventricle.

Auscultation of the apical area reveals a marked diminution in the muscular element of the first sound of the heart which is brief, of high pitch, and of a valvular quality closely approaching that of the normal second sound of the heart. Similarly the aortic second sound at the right second interspace is diminished in intensity, in striking contrast to the coincident accentuation of the second sound at the pulmonic area. A soft blowing systolic murmur of relative mitral regurgitation is frequently audible at the apex and the aortic area is apt to be the site of a systolic or diastolic murmur due to disease of the aortic valve. Reduplication of the first or second sound of the heart is frequently encountered and the gallop-rhythm of Potain is occasionally to be elicited.

Right Ventricular Dilatation.—*Inspection* of the precordia in the presence of right ventricular dilatation usually reveals a very diffuse and undulatory cardiac impulse, which occupies a position along the left sternal border, in the upper epigastrium, or extending to the right of the sternum.

Palpation reveals the slapping character of the contraction of the dilated ventricle, which is in marked contrast to the powerful thrusting movement of the chamber in the presence of right ventricular hypertrophy. Bimanual palpation of the liver at this time will frequently elicit a systolic pulsation of this organ, occurring as the result of tricuspid regurgitation.

Upon *percussion* it is usually possible to demonstrate a minor displacement of the cardiac outline toward the left but not downward; but the principal alteration in the cardiac outline is extension of the dullness far to the right of the sternum, where it encroaches upon the normal pulmonary resonance of Ebstein's cardiohepatic angle.

Percussion of the pulmonary bases usually elicits dullness due to pulmonary edema; and delimitation of the areas of hepatic and splenic dullness reveals an extension of dullness due to chronic passive congestion of these organs.

Auscultation reveals enfeeblement of both sounds of the heart at the apex with diminution in the muscular element of the first sound, which approaches the valvular quality of the normal second sound of the heart. The pulmonic second sound at the base is markedly enfeebled. The soft and blowing murmur of relative tricuspid insufficiency is frequently audible at the tricuspid area.

Auricular Dilatation.—Dilatation of the left auricle affords few physical manifestations of its presence owing to the deep location of this chamber of the heart. It may be possible to demonstrate an extension of cardiac dullness to the left in the second left intercostal space, and a visible impulse is occasionally perceptible in the same area.

Right auricular dilatation is manifested by extension of the area of cardiac dullness toward the right in the second and third right intercostal spaces, and a visible pulsation in the same area.

Diagnosis.—Cardiac dilatation is readily recognized by the character of the heart sounds, the increased extent of the area of cardiac dullness, the diffuse and undulatory cardiac impulse, which is displaced from its normal site, and the signs of systemic venous engorgement.

Differential Diagnosis.—The enlarged heart of *cardiac hypertrophy* is differentiated from cardiac dilatation by the strong and heaving cardiac impulse, which is clearly visible and is displaced from its normal site, the accentuated second sounds of the heart, the full and regular pulse, and the absence of signs of venous engorgement.

The differential points between cardiac dilatation and *pericarditis with effusion* have been described under the latter disease.

Increased dullness to the left of the heart which is due to *consolidation of the left lung* is differentiated by the presence of bronchial breathing and râles over the area of pulmonary disease.

The area of cardiac dullness may also be extended by crowding forward of the heart by the growth of a mediastinal tumor; but these tumors produce pressure symptoms which are more pronounced than any which are produced by enlargement of the heart.

Encysted pleurisy may be confused with cardiac dilatation by broadening the area of dullness around and adjacent to the heart; but here there is usually the friction sound, and the heart sounds are not altered, and there are no signs of venous engorgement as in the case of cardiac failure.

The presence of hypertrophic emphysema, by the interposition of the anterior borders of the lungs between the heart and chest wall, may effectually mask the cardiac enlargement, and require careful percussion to bring out the increase in the size of the organ.

CONGENITAL HEART DISEASE

Clinical Pathology.—Pulmonary stenosis is the most frequent and clinically the most important of the congenital lesions of t heart. The stenosis may be complete, the orifice of the vessel being closed by a fibrous membrane, or may permit all gradations of patency. In addition to obstruction at the valve, there may be narrowing of the conus arteriosus of the right ventricle, or the pulmonary artery may be congenitally narrow beyond the valvular opening.

The second most common congenital lesion of the heart is the patent foramen ovale, which normally closes during the first week of extrauterine life, but which may remain partially open to adult life in fourteen per cent of persons.

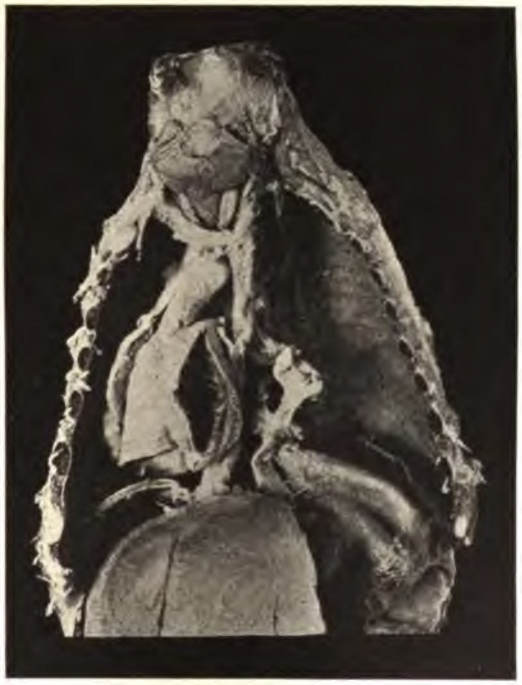


Fig. 188.—Reptilian heart. (From Delafield and Prudden.)

The interauricular septum may be found absent, resulting in the reptilian heart or cor triloculare. In other instances both the interauricular and interventricular septa are absent, the heart consisting of only two chambers, the cor biloculare.

In a certain number of cases the ductus arteriosus, which usually closes during the first month of extrauterine life, remains patent t [illegible] to signs of congenital heart disease.

Co [illegible] of the aortic, mitral, and tricuspid valves are infrequently encountered. At the various valves of the heart, there may be supernumerary cusps or a diminution of the number of cusps, or adhesions between them, or merely a button-hole slit in a membrane closing an orifice.

In certain unusual cases the aorta is found to arise from the right ventricle and the pulmonary artery from the left ventricle; while in cases of visceral transposition the heart lies chiefly in the right half of the thorax.

Physical Signs.—The physical signs of congenital heart disease are early apparent, the most striking sign being extreme blueness or cyanosis of the child. However, cyanosis may be absent in the presence of cardiac disease. The finger-tips are often clubbed, the so-called Hippocratic fingers. Dyspnea is invariably present.

As pulmonary stenosis is the most frequently underlying lesion, there is in most cases a systolic blowing murmur to the left of the sternum in the pulmonic area, with signs of hypertrophy of the right ventricle. On the other hand, a patent ductus arteriosus yields a rather prolonged, systolic murmur in the same area which is, however, more distinctly audible in the third left interspace.

Diagnosis.—A diagnosis of congenital heart disease can be readily made in many cases on the extreme cyanosis, dyspnea, clubbed fingers, and loud blowing murmurs. However, it is often very difficult to say with certainty just what the underlying lesion is, as the signs are often confusing and frequently two conditions coexist.

The murmurs of congenital disease are very difficult to differentiate from functional murmurs in anemic children; but it should be borne in mind in this connection that functional murmurs are not transmitted and do not produce alterations in the myocardium. From acquired heart disease, congenital disease is usually differentiated by the fact that it is present from birth, the child from birth having been blue (morbus cæruleus); that the child is usually under two years of age, at which time acquired lesions are practically unknown; and that the murmurs are atypical in location and transmission.

ANEURYSM OF THE AORTA

Clinical Pathology.—In the induction of aneurysm of the aorta two factors are interactive to produce the dilatation and partial rupture of the arterial coats; namely, disease of the vessel wall in the form of atheroma, and strain exerted upon the vessel wall through hypertension in the greater circulation. Disease of the vessel wall arises through the prolonged action of the toxins of the acute infectious fevers, gout, and chronic mineral intoxications, and preeminent among these predisposing factors ranks syphilis.

The disease is most apt to attack male subjects during the third and fourth decades of life, the age at which arterial pressure is apt to be heightened by laborious occupations and when beginning atheroma is not infrequent. The disease is prone to develop in persons following laborious occupations in which the heart is repeatedly subjected to strain.

Aortic aneurysm is most frequently a solitary lesion, although multiple aneurysmal dilatations of the vessel are encountered. The dilatation may assume the form of a fusiform enlargement of the lumen of the vessel, or it may take the form of a saccular dilatation. The site of solitary aneurysm may be the ascending aortic arch, the transverse arch of the aorta, the descending aortic arch, the descending thoracic aorta, or the abdominal aorta.

The saccular aneurysm of the ascending arch of the aorta usually develops upon the convexity of the arch above the pericardium and extends toward the right and forward, reaching the anterior chest wall in the second or third right intercostal space. In this situation the aneurysm may cause compression of the superior vena cava with resulting engorgement of the cervical veins, or it may compress the subclavian vein alone, producing venous stasis and edema of the right arm. With less frequency the aneurysm develops upon the concavity of the ascending aorta, producing an area of dullness along the right sternal border, and occasionally the aneurysm is situated in the proximal portion of this part of the aorta and is enveloped by the pericardium. Aneurysm of this portion of the aorta displaces the heart downward and toward the left; and the usual termination of the disease is by rupture into the pleural cavity, the pericardium, or rarely into the superior vena cava.

Aneurysm of the transverse arch of the aorta in the majority of cases springs from the posterior aspect of the vessel, with less frequency from the anterior wall, and with least frequency from the concavity of the arch. Posterior aneurysm of the aortic arch extends backward toward the vertebral column, resulting in pressure symptoms arising from compression of the trachea and esophagus, and producing pupillary disturbances and paroxysmal cough from compression of the recurrent laryngeal nerve, which hooks around this portion of the vessel. Aneurysm springing from the anterior wall of the aortic arch progresses forward and erodes the sternum, causing a tumor which points toward the right sternal margin more often than toward the left of the median line. Aneurysm springing from the concavity of the arch progresses downward, with compression of the left bronchus, and recurrent laryngeal nerve disturbances.

Aneurysm of the descending aortic arch usually springs from the posterior aspect of the vessel and progresses backward and toward the left, causing erosion of the thoracic vertebræ between the third and sixth dorsal segments. Pressure is exerted upon the roots of the spinal nerves, the left bronchus, or the root of the lung; and rupture may occur into the left bronchus, the trachea, or the esophagus.

Aneurysm of the descending thoracic aorta usually springs from the inferior portion of the vessel, just above the diaphragm, and the sac develops in contact with the lower thoracic vertebræ. The bodies of the lower thoracic vertebræ are eroded and dysphagia is a common symptom from compression of the esophagus.

Aneurysm of the abdominal aorta, which is relatively less frequently encountered than is aneurysm of the thoracic portion of the vessel, usually springs from the ventral aspect of the aorta immediately below the diaphragm in the vicinity of the origin of the celiac axis, whence the tumor progresses forward to produce bulging of the epigastrium or left hypochondriac region. With less frequency the aneurysm takes origin from the posterior aspect of the abdominal aorta and progresses backward and erodes the bodies of the vertebræ. Rupture may occur into the retroperitoneal tissues, or into the peritoneal cavity, and rarely a communication is established with the inferior vena cava.

The course of the disease is insidious and progressive, resulting in rupture of the sac in 75 per cent of the cases. In some instances the disease remains latent for a prolonged period, but with the ever-increasing dimensions of the tumor, pressure symptoms eventually come into the foreground of the clinical picture. Of these dyspnea is often one of the most urgent. This occurs from compression of the right heart, the trachea, bronchi, pulmonary artery or vagus nerve, as the case may prove. Cough is frequently excessive arising partially as a result of recurrent laryngeal nerve compression, and frequently from interference with the pulmonary circulation with the induction of chronic bronchial inflammation. Dysphagia is apt to arise from compression of the esophagus or from compression of the recurrent laryngeal nerve producing spasm of the esophagus. Erosion of the sternum or of the bodies of the vertebræ is attended by pain of a dull and boring character, and compression of the vagus nerves is followed by pseudoanginal attacks.

Physical Signs.—*Inspection.*—In the presence of aneurysm of the ascending or transverse arch of the aorta inspection of the anterior surface of the thorax by oblique illumination frequently reveals the presence of a systolic pulsation in the second and third intercostal spaces adjacent to the sternum or in the suprasternal notch. In the presence of aneurysm of the descending aortic arch a similar pulsation is frequently to be encountered in the left interscapular region at the level of the scapular spine. In advanced cases of aneurysm of the arch there may be a visible protrusion of the thoracic wall in the region of the manubrium sterni which exhibits true expansile pulsation during ventricular systole. The cardiac impulse frequently occupies a position lower and farther to the left than in the normal subject, as a result of displacement of the heart by the growth of the aneurysm.

The tracheal tug of Oliver may be elicited in many cases of aneurysm of the aortic arch and should always be sought for by the examiner.

Palpation.—Palpation with the palm of the hand gently applied to the pulsating area is useful in demonstrating true expansile pulsation and in estimating the resistance which is offered by the underlying structures to the impact of the blood stream. Frequently the wall covering the pulsation is quite resistant, while in other instances of perforating aneurysm the pulsating area is soft and readily compressible.

The *pulse* in aortic aneurysm is of service in estimating the location of the lesion. In the presence of aneurysm of the ascending aortic arch there is no asymmetry in the two radial pulses. When, however, the aneurysm involves the arch distal to the origin of the innominate artery, the left radial pulse is delayed, resulting in asymmetry of the radial pulse upon the two sides of the body.

Percussion.—In the presence of extensive aneurysmal dilatation of the ascending aortic arch there is a demonstrable area of dullness extending to the right of the area of vascular dullness in the second and third intercostal spaces to the right of the sternum. In the cases of aneurysm of the transverse arch there is an area of dullness extending to the right and to the left of the manubrium sterni; and in the presence of aneurysm of the descending arch there is a demonstrable area of dullness in the left interscapular region at the level of the spine of the scapula. Small aneurysmal dilatations of the aorta do not, however, appreciably modify the percussion note of the thorax.

Auscultation.—Auscultation of the thorax overlying an aortic aneurysm is apt to reveal the presence of a systolic murmur; but the detection of a murmur is not sufficient ground for a diagnosis of aneurysm. More important than the murmur in a suspected case is the presence of a loud, ringing and accentuated aortic second sound, a sign which is rarely absent in the presence of aneurysm of the aortic arch. Occasionally this accentuated sound is found replaced by a diastolic murmur at the aortic valve in cases which are unquestionably aneurysmal. A systolic murmur is frequently appreciable in the carotids in the presence of aneurysm of the arch, but a similar transmitted murmur attends organic stenosis at the aortic valve.

The symptoms and physical signs emanating from aneurysm of the abdominal aorta are in no wise as distinctive as is the case with aneurysm of the thoracic aorta. There is usually dull and boring pain and frequently a visible pulsation of the epigastric region. When such a pulsation is encountered the patient should be examined in the knee-chest posture in order to cause any intra-abdominal tumor to fall away from the subjacent aorta. Care should also be taken to exclude the systolic pulsation of the epigastrium which attends massive right ventricular hypertrophy.

Diagnosis.—The cardinal signs of aortic aneurysm comprise dullness at the base of the heart, extending to the right or left of the sternum, dullness in the left interscapular region at the level of the scapular spine, pupillary changes, and pressure symptoms, occurring in a subject of a laborious occupation or suffering with syphilis. Later in the evolution of the disease boring pain becomes a marked feature of the case with cough, cardiac disturbances, asymmetry of the pulses and visible tumor with true expansile pulsation. Fluoroscopy is of aid in establishing the diagnosis and in localizing the site of the lesion.

PART II. THE ABDOMEN

SECTION I

GENERAL EXAMINATION OF THE ABDOMEN

CHAPTER XXI

CLINICAL ANATOMY OF THE ABDOMEN

The abdomen, the portion of the trunk which is limited upon the surface of the body by the ensiform cartilage and the costal arch superiorly and by the pubic crest and Poupart's ligaments inferiorly, presents an irregularly oval contour, the shape varying, however, with the age and the sex of the subject. Thus, the abdomen of the child is roughly conical with the apex directed inferiorly; while in the adult female subject, owing to the unusual breadth of the pelvis in this sex, it is roughly conical with the apex directed superiorly; whereas in the case of the adult male subject the abdomen is oval or barrel-shaped, with a moderate anteroposterior flattening.

The *abdominal cavity* is limited superiorly by the inferior aspect of the diaphragm, and inferiorly by the levator ani, assisted by the coccygeus, these two muscles constituting the pelvic diaphragm. The more roomy upper portion of the abdominal cavity, situated above the brim of the pelvis, is termed the *abdomen proper,* while the smaller portion of the cavity situated below the pelvic inlet is termed the *pelvis.*

The abdominal cavity is not limited superiorly by the lower border of the costal arch, which forms its upper boundary upon the surface of the abdomen, but by the vault of the diaphragm, extending upward into the bony thorax for a considerable distance. Upon the right side its superior limit is on a level with the upper border of the fifth rib in the midclavicular line; while upon the left side of the body its superior limit is approximately one-half inch lower in the same line.

The *abdominal wall* is composed largely of muscular and **soft** structures, reinforced in certain regions by bony structures. Anteriorly and laterally the wall is formed of the abdominal muscles, the lower ribs and iliac bones. Posteriorly it is formed by the muscles of the posterior abdominal wall, the quadratus lumborum and psoas upon either side, and in the median line by the vertebral column. The anterior abdominal wall and the lateral walls between the last rib and the iliac crest are devoid of bony support and are subject to distention and retraction, depending upon the state of the abdominal contents.

Within the abdominal cavity the liver occupies the upper right quadrant of the cavity, sheltered largely beneath the inferior right costal margin, but extending also into the subcostal angle in the epigastric region. The spleen occupies a deep position in the upper left quadrant, sheltered by the ninth, tenth, and eleventh ribs and in apposition with the fundus of the stomach. The stomach occupies a position in the superior portion of the abdominal cavity, between the liver and the spleen; and from the greater curvature of the stomach the great omentum descends for a variable distance and covers the coils of the small intestine. The coils of the small intestine occupy the central and inferior portions of the abdominal cavity, slightly overlapping the ascending and descending colon in the lumbar regions. The large intestine envelops the small intestine in the form of a frame with an inferior concavity upon three sides of the abdominal cavity. The urinary bladder occupies the anterior portion of the pelvis; and, if it is in a state of distention, rises above the pubic crest to mount into the hypogastric region. The rectum occupies the posterior portion of the pelvis, lying in the concavity of the sacrum.

ANATOMICAL LANDMARKS OF THE ABDOMEN

At the superior limit of the anterior abdominal surface in the median line is the *ensiform cartilage*, with the *costal arch* descending from it upon either side of the abdomen. The anterior extremities of the fifth, sixth, seventh, eighth, ninth, and tenth costal cartilages are palpable; and, in the case of thin subjects with relaxation of the abdominal walls, the free extremities of the eleventh and twelfth ribs as well may be palpated.

At the inferior limit of the abdomen the *symphysis pubis* with its *pubic spines* is encountered, and extending from them in a direction outward and obliquely upward, one encounters *Poupart's ligament* upon either side of the abdomen.

In the inferior and lateral regions the *iliac crest*, terminating anteriorly in the *anterior superior iliac spine* is encountered, the latter remaining plainly palpable even in obese subjects.

In the lower central region of the abdominal surface the *umbili-*

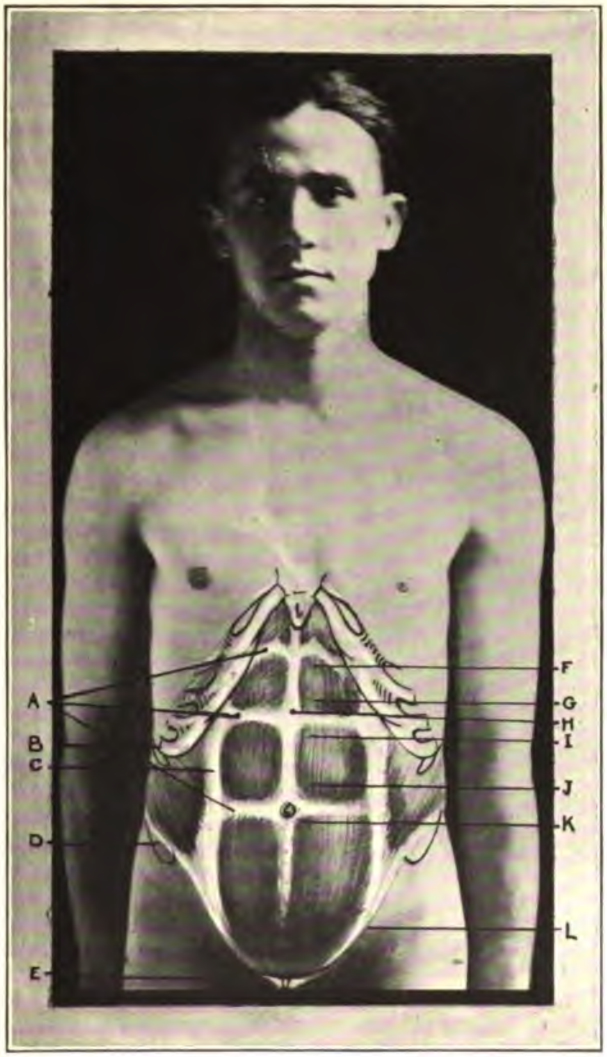


Fig. 189.—Anatomical landmarks of abdomen:

A. Lineae transversae. B. Costal arch. C. Linea semilunaris. D. Anterior superior iliac spine. E. Symphysis pubis. F. Commencement of abdominal aorta. G. Origin of celiac axis. H. Linea alba. I. Origin of superior mesenteric artery. J. Origin of inferior mesenteric artery. K. Bifurcation of aorta. L. Poupart's ligament.

cus is noted. It corresponds to the level of the disc between the third and fourth lumbar vertebræ.

The *linea alba* extends in the median line from the ensiform cartilage to the symphysis pubis. It is indicated by a slight groove in the median line of the abdomen above the level of the umbilicus, and by a line of hair or of brown pigment, the *linea nigra,* below the umbilicus.

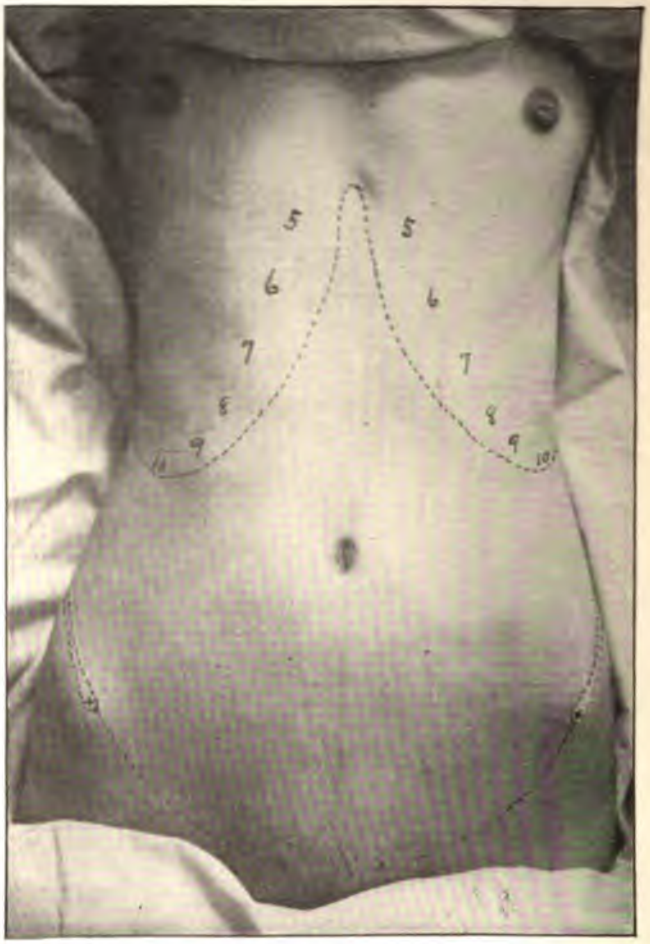


Fig. 190.—The abdominal surface with the rib margins and the iliac crests outlined. (From Crossen.)

The *linea semilunaris,* upon either side of the abdomen, extends with a slight convexity outward from the junction of the tip of the ninth costal cartilage with the external border of the rectus muscle to the pubic spine. It corresponds accurately to the external limit of the sheath of the rectus muscle.

Lineæ transversæ are to be noted in subjects of good muscular development. They are transverse constrictions in the rectus muscles. They are three in number, as a rule, although a fourth may sometimes be encountered. One constriction is located at the level of the ensiform cartilage, another at the level of the umbilicus, and a third constriction midway between these two. When a fourth constriction is present, it is located midway between the umbilicus and the symphysis pubis.

Cutaneous flexion folds are encountered in obese subjects. They are usually two in number, one at the level of the umbilicus and the other just above the symphysis pubis.

TOPOGRAPHICAL ANATOMY

The course of the *abdominal aorta* corresponds to a vertical line upon the anterior surface of the abdomen extending from a point a little to the left of the ensiform process downward to a point three-fourths of an inch below and a little to the left of the umbilicus, where the vessel bifurcates to form the common iliac arteries. Upon this line the celiac axis arises from the abdominal aorta at a point four and one-half to five inches above the umbilicus; the superior mesenteric artery arises at a point four inches above the umbilicus; the renal artery arises three and one-half inches above the umbilicus; and the inferior mesenteric artery arises one inch above the level of the umbilicus.

The course of the *common iliac* and *external iliac* arteries corresponds to a line drawn from the point of bifurcation of the abdominal aorta to a point midway between the anterior superior iliac spine and the symphysis pubis.

The course of the *deep epigastric* artery is represented by a line drawn from the midpoint of Poupart's ligament upward and inward to the umbilicus.

The course of the *inferior vena cava* is represented by a vertical line drawn along the line representing the course of the abdominal aorta, a little distance to the right side of this line.

The *common* and *external iliac* veins are indicated by lines upon the abdominal surface slightly below and to the right of and corresponding in direction to the lines of the arteries of the same name.

The topographical anatomy of the various abdominal organs is described in the sections dealing with the respective organs.

cavity is divided into nine regions. The *epigastric region,* bounded inferiorly by the subcostal line, and superiorly and laterally by the line of the costal arch, overlies the stomach, duodenum, liver, gall bladder, pancreas, and portions of the two kidneys.

The *left hypochondriac* region, limited inferiorly by the subcostal line and internally by the line of the left costal arch, overlies the fundus of the stomach, the spleen, and the splenic flexure of the colon.

The *right hypochondriac* region, limited inferiorly by the subcostal line and internally by the line of the right costal arch, overlies the portion of the abdominal cavity which is occupied by the liver and right kidney.

The *umbilical region,* limited superiorly by the subcostal line, inferiorly by the intertubercular line, and laterally by the right and left mid-Poupart lines, overlies the coils of the small intestine, the mesentery, the great omentum, a portion of the two kidneys, and of the transverse colon.

The *left lumbar region,* bounded superiorly by the subcostal line, inferiorly by the intertubercular line, and internally by the left mid-Poupart line, overlies the left kidney, the descending colon, and some coils of the small intestine.

The *right lumbar region,* limited superiorly by the subcostal line, inferiorly by the intertubercular line, and internally by the right mid-Poupart line, overlies a portion of the right kidney, of the ascending colon, and coils of the small intestine.

The *hypogastric region,* lying below the intertubercular line and limited laterally by the mid-Poupart lines, and inferiorly by the pubic symphysis, overlies the distended bladder, some coils of the small intestine, a portion of the sigmoid flexure, the cecum, occasionally the vermiform appendix, and the pregnant uterus.

The *left iliac region,* limited superiorly by the intertubercular line, inferiorly by Poupart's ligament, and internally by the left mid-Poupart line, overlies the sigmoid flexure of the colon.

The *right iliac region,* limited by the intertubercular line, the right mid-Poupart line, and Poupart's ligament, overlies the cecum and usually the vermiform appendix.

Instead of the division of the abdominal cavity into nine regions by means of two horizontal and two vertical lines, the cavity may be divided into four regions, or quadrants, through the medium of a horizontal and a vertical line passing through the umbilicus. In this method of subdivision the four regions are termed, respectively, the right upper quadrant, the left upper quadrant, the right lower quadrant, and the left lower quadrant, of the abdomen.

The *right upper quadrant* of the abdomen overlies the right lobe of the liver with the gall bladder, the hepatic flexure and the proximal portion of the transverse colon, the pylorus, the first and second portions of the duodenum and the head of the pancreas, and more deeply in this region, the superior half of the right kidney.

The *left upper quadrant* of the abdomen overlies the left lobe of the liver, the stomach, the distal half of the transverse colon with the splenic flexure, the larger portion of the pancreas, the superior half of the left kidney, and the spleen.

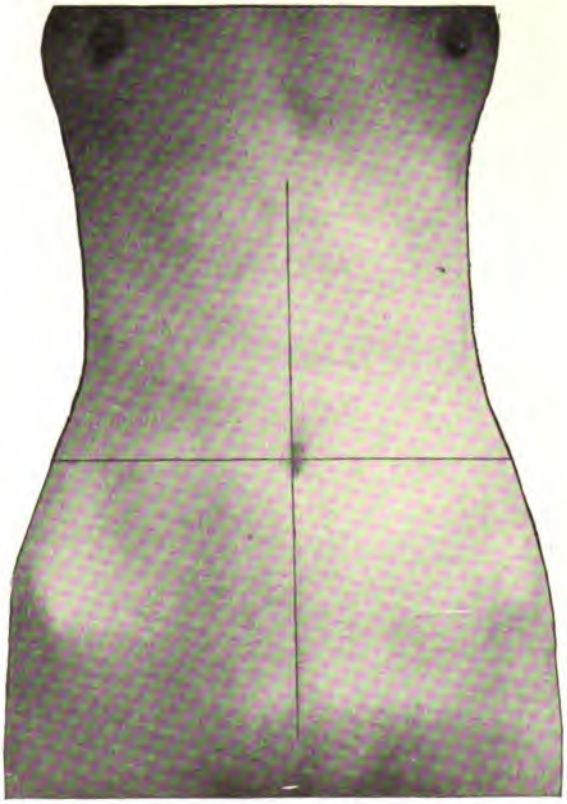


Fig. 193.—The abdominal surface divided into quadrants. (From Crossen.)

The *right lower quadrant* of the abdomen overlies the cecum with the vermiform appendix, the ascending colon, the inferior half of the right kidney, the right fallopian tube and ovary, together with portions of the bladder and the uterus.

The *left lower quadrant* of the abdomen overlies the descending colon and sigmoid flexure, a portion of the inferior half of the left kidney, coils of the small intestine, the left fallopian tube and ovary, and a portion of the bladder and of the uterus.

A very convenient subdivision of the abdominal cavity is that devised by Crossen, in which the natural landmarks of the abdomen are utilized, the only artificial lines employed being one encircling the umbilicus, and a horizontal line drawn from either side of the circle. By this method the abdomen is subdivided into regions which are respectively designated a right upper, left upper, central upper, right lower, left lower, central lower, umbilical right lumbar, and left lumbar.

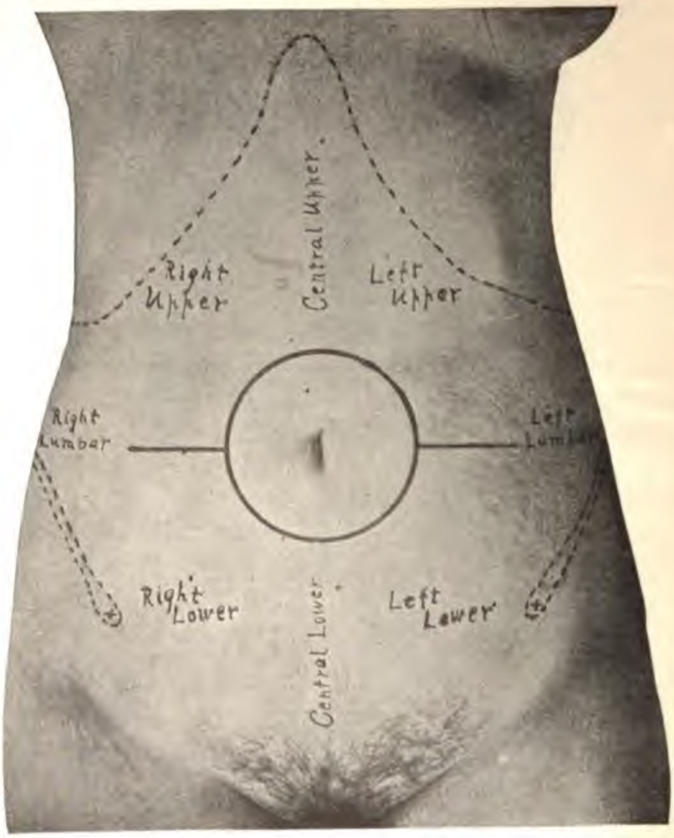


Fig. 194.—Another abdomen divided with the circle and short horizontal lines, and showing the names on the primary regions. The area within the circle carries the usual designation, "umbilical region." (From Crossen.)

CHAPTER XXII

INSPECTION OF THE ABDOMEN

Technic.—During inspection of the abdomen the patient should in the first instance assume the dorsal decubitus, the position in bed or upon the examining table being absolutely symmetrical and free from undue tension of the abdominal muscles. The subject should be covered with a sheet which may be turned down, exposing the abdomen freely to a short distance above the pubic symphysis. The examiner should in the first place stand beside the abdomen and inspect its surface first by direct illumination and afterward by oblique illumination, the latter frequently revealing slight pulsations, protrusions or vermicular movements which escaped detection during the examination by direct illumination. The examiner should then assume, consecutively, positions near the feet and near the head of the subject and should inspect the abdominal wall from these two positions.

For the purpose of detecting certain phenomena such as the vermicular movements of visible peristalsis and abdominal sagging due to visceroptosis, the abdomen should in addition be inspected with the subject in the standing posture. Finally, in certain instances it is desirable to examine the patient in the genupectoral posture, a position which permits movable tumors of the abdominal viscera to fall forward and to become more clearly visible upon inspection of the abdominal surface.

THE SKIN OF THE ABDOMEN

The integument of the abdomen is lax and loose in the presence of emaciation from chronic wasting disease, and is tense and glistening in the presence of abdominal distention from ascites, pregnancy, or increased intraabdominal tension attending the development of a large intraabdominal tumor. Whitish or silvery stridæ, *lineæ albicantes,* distributed over the lower portion of the abdomen and the upper portions of the thighs are indicative of former abdominal distention, whether from tumor, ascites, or pregnancy.

Scars upon the abdomen may result from former surgical operations, from trauma, or from the eruption of syphilis or other cutaneous disease. A scar in the groin is suggestive of suppuration of an inguinal gland, which has opened spontaneously or has been opened by the surgeon.

The *linea nigra,* a line of brown pigment in the median line of the abdomen below the umbilicus, accompanies pregnancy and chronic abdominal distention from other cause.

The abdomen is the usual site of the cutaneous eruption of typhoid fever, the *rose spot* of this disease. These spots are small, discrete, hyperemic, slightly elevated papules, which readily disappear upon pressure. In certain instances the papule is surmounted by a small vesicle. They are not limited to the abdomen, though most frequently encountered in this locality; but may appear upon the back, the arms, or the thighs. The rose spots appear in successive crops, disappearing in two or three days, sometimes leaving a brownish stain.

During pregnancy the abdominal skin occasionally exhibits the brownish areas of chloasma.

ENLARGEMENT OF THE SUPERFICIAL VEINS OF THE ABDOMEN

In the presence of portal obstruction, there is present upon the abdominal wall a series of distended superficial veins extending outward from the umbilicus in a radial manner, constituting the *caput medusæ.* A distention of the superficial veins over the abdomen, communicating with similarly distended veins over the thorax, is indicative of portal vein obstruction by hepatic cirrhosis or tumor, chronic ascites, or pressure exercised upon the inferior or superior vena cava by abdominal or mediastinal tumor. The direction of the blood current in the distended vein is an index to the site of the obstruction. If, upon compression of the vein, it is found that the direction of the blood current is upward, the obstruction is in the portal vein or inferior vena cava; whereas, if the current is directed downward, the obstruction is situated in the course of the superior vena cava. The venous tortuosity under these circumstances is an evidence of the effort at the establishment of collateral circulation in the presence of obstruction of the usual venous channels.

THE UMBILICUS

The umbilicus should be examined for protrusion, retraction, skin eruptions, and inflammation. A protruding umbilicus i noted in umbilical hernia, during the latter months of pregnancy in the presence of portal vein obstruction, ascites, or abdomina distention due to the development of a large intraabdomina tumor. In the obese subject on the contrary the umbilicus i markedly retracted.

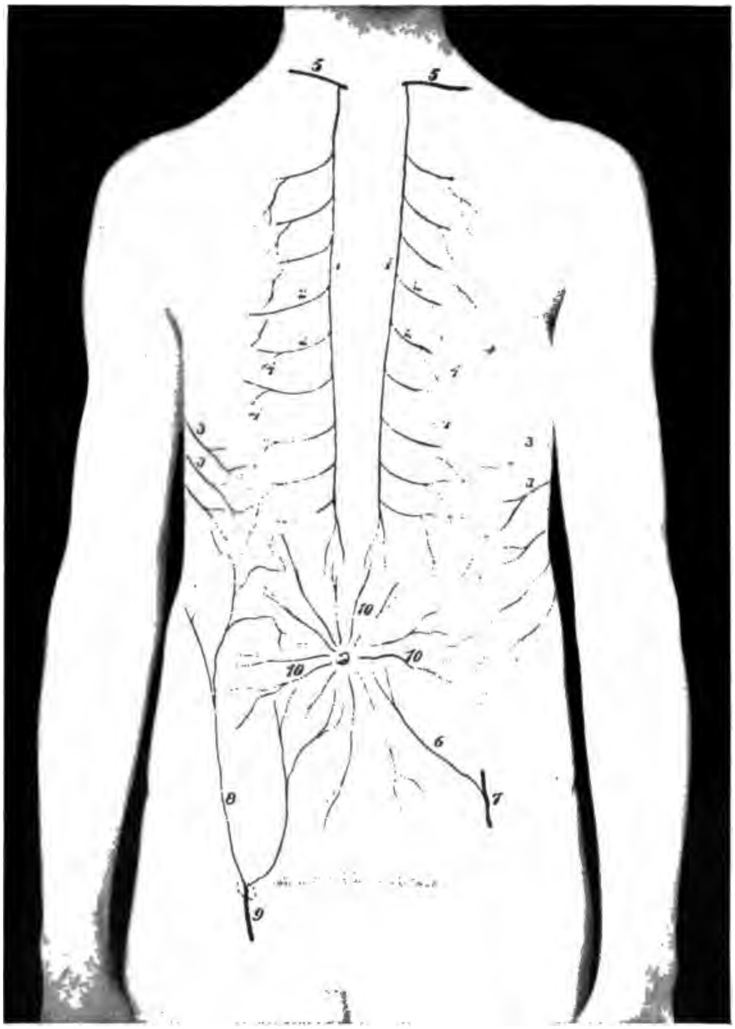


Fig. 195. —Establishment of collateral circulation in portal vein obstruction and media tinal tumor. (Eisendrath.)

1, internal mammary veins; *2*, anterior intercostal veins; *3*, posterior intercostal vein *4*, radicles of *2*; 5, subclavian vein; *6*, deep epigastric vein; *7*, external iliac vein; *8*, sup ficial circumflex iliac vein; *9*, internal saphenous vein; *10*, caput medusae.

ENLARGED GLANDS

The glands in the groin are enlarged as a result of venereal infection or localized nonspecific inflammation resulting from abrasions about the external genitalia or of the lower extremities. The character of the adenopathy whether hard and discrete or soft and fluctuating with matting of the glands, should be de-

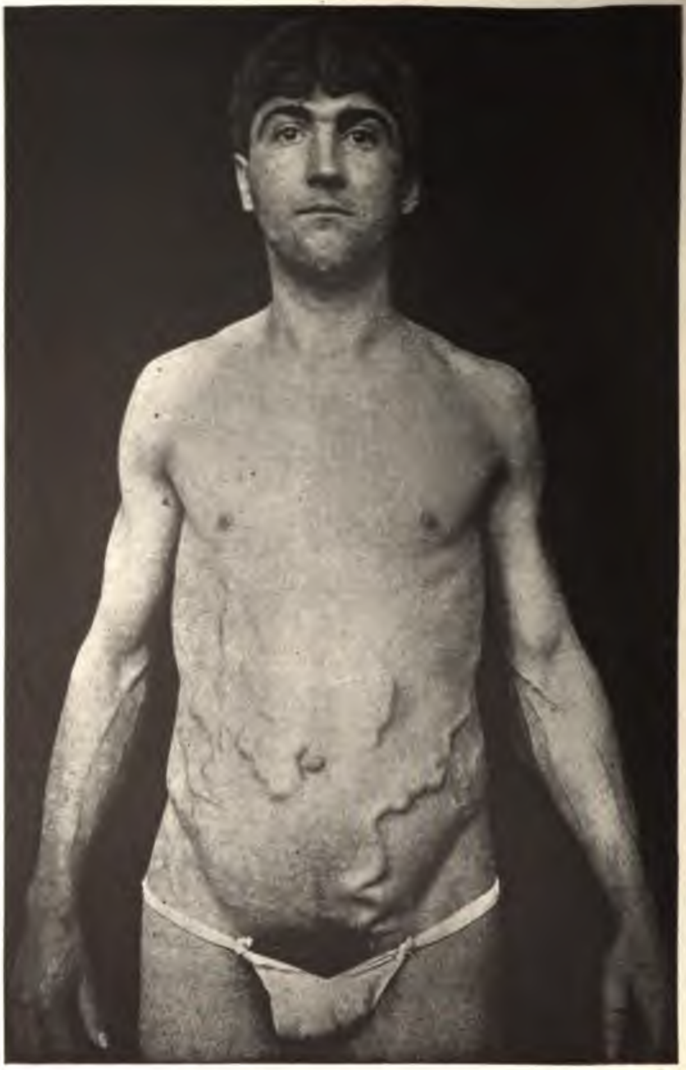


Fig. 196.—Abdominal arteries in a case of double iliac thrombosis of typhoid origin. (Woolley, after Thayer.)

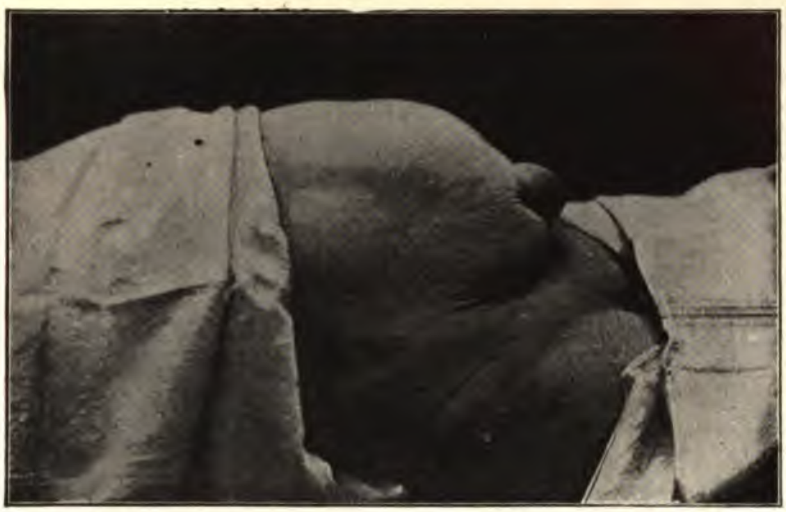


Fig. 197.—A small umbilical hernia, with a relaxed abdominal wall. (Crossen, after Hirst.)

termined with the view of the differentiation of luetic and chancroidal adenopathies.

VISIBLE PERISTALSIS

The peristaltic movements of the stomach, small intestine, or large intestine at times become visible as a vermicular movement upon the anterior abdominal wall. In its exaggerated form it is indicative of obstruction located at the pylorus, at some point in the course of the small intestine, or in the colon.

The peristaltic movement of the stomach is visible under these circumstances in the upper portion of the abdomen, pursuing a

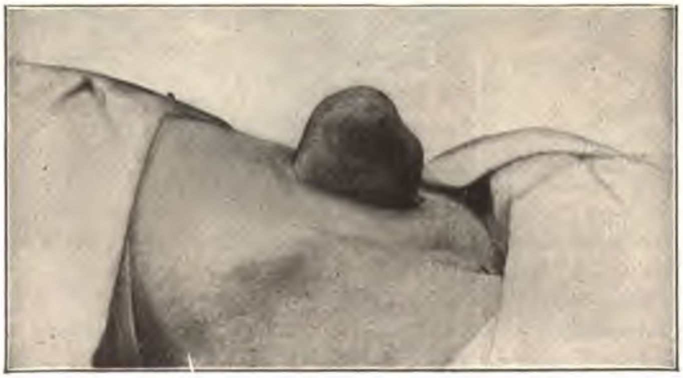


Fig. 198.—A large ventral hernia at the site of an operation scar. (Crossen, after Hirst.)

direction from left to right and somewhat downward in the epigastric region.

Visible peristalsis of the small intestine is chiefly confined to the umbilical region, whereas that occasioned by obstruction of the large intestine is observed over the course of the colon. When it is not unduly pronounced, peristaltic movement of the abdominal wall may often be accentuated by applying a cold hand to the abdominal surface or by flicking the surface of the abdomen with a towel which has been wet in cold water. When the

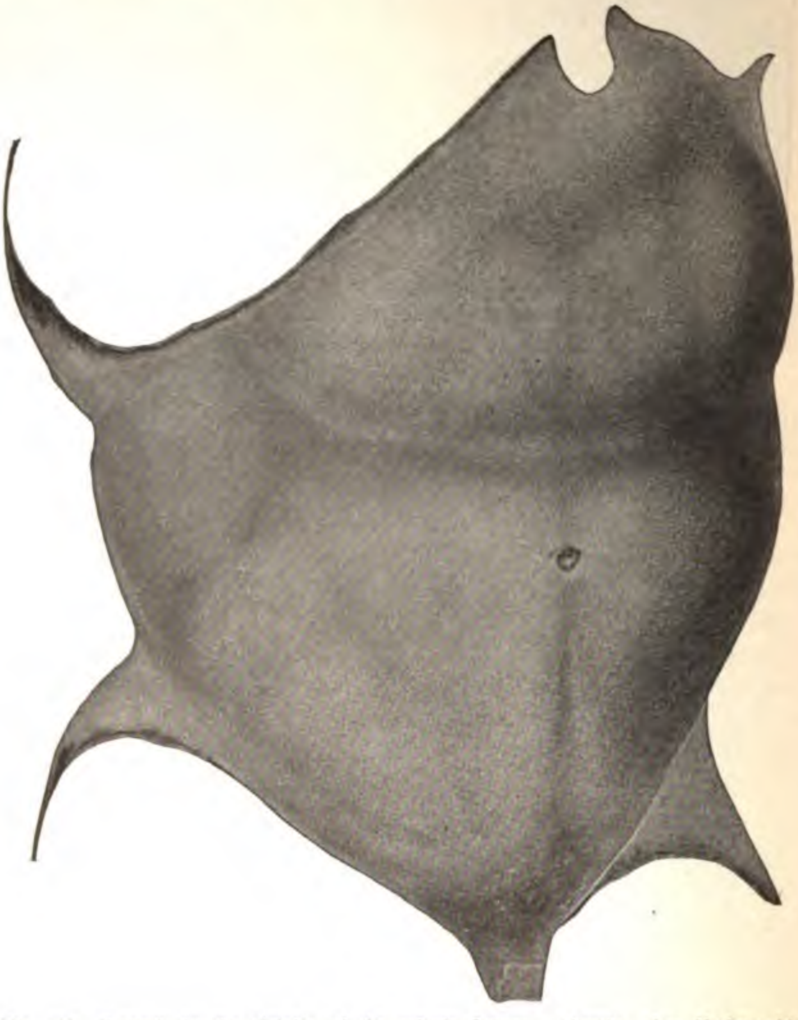


Fig. 199.—Stenosis in the vicinity of the splenic flexure. (Austin, after Nothnagel.)

site of the obstruction is situated in the ileum just proximal to the ileocecal valve, the visible peristalsis assumes frequently a "ladder pattern," the waves lying one above the other in the umbilical region.

Visible peristalsis observed in extremely emaciated patients with extremely thin abdominal walls, or in women in whom repeated pregnancy has caused a diastasis of the rectus muscles, possesses no diagnostic significance.

ABOLITION OF THE RESPIRATORY MOVEMENTS OF THE ABDOMEN

Fixation of the abdominal wall with inhibition or abolition of the respiratory movements is indicative of pain arising within the abdominal cavity with the respiratory excursion of its walls, which is not infrequently due to acute peritonitis.

VARIATIONS IN THE CONTOUR OF THE ABDOMEN

The normal abdomen is symmetrical with a moderate degree of anteroposterior flattening in the male subject. The walls are of uniform tension, and it presents a moderate bulging of the inferior regions in the female subject. The umbilicus of the normal abdomen is neither unduly depressed nor protruding.

Variations in the contour of the abdomen may be symmetrical or asymmetrical. Symmetrical enlargement of the abdomen is encountered in the presence of obesity, pregnancy, intestinal meteorism, ascites, and in the presence of visceroptosis. Symmetrical retraction of the abdomen is encountered in conditions of emaciation depending upon cardiac or pyloric stenosis, or the moribund state.

OBESITY

In the obese subject the abdomen is symmetrically enlarged; the cutaneous flexion folds are accentuated; the umbilicus is unduly depressed; and the inferior regions of the abdomen are pendulous, encroaching to a variable extent upon the thighs and the pubes.

PREGNANCY

The abdominal enlargement accompanying pregnancy is progressive, increasing gradually as the uterus rises out of the pelvis, and comes to occupy the abdominal cavity. In its fully developed state the umbilicus protrudes, the abdominal skin presents the lineæ albicantes of pregnancy, and the abdominal distention is accompanied by changes in the breasts and the positive signs of

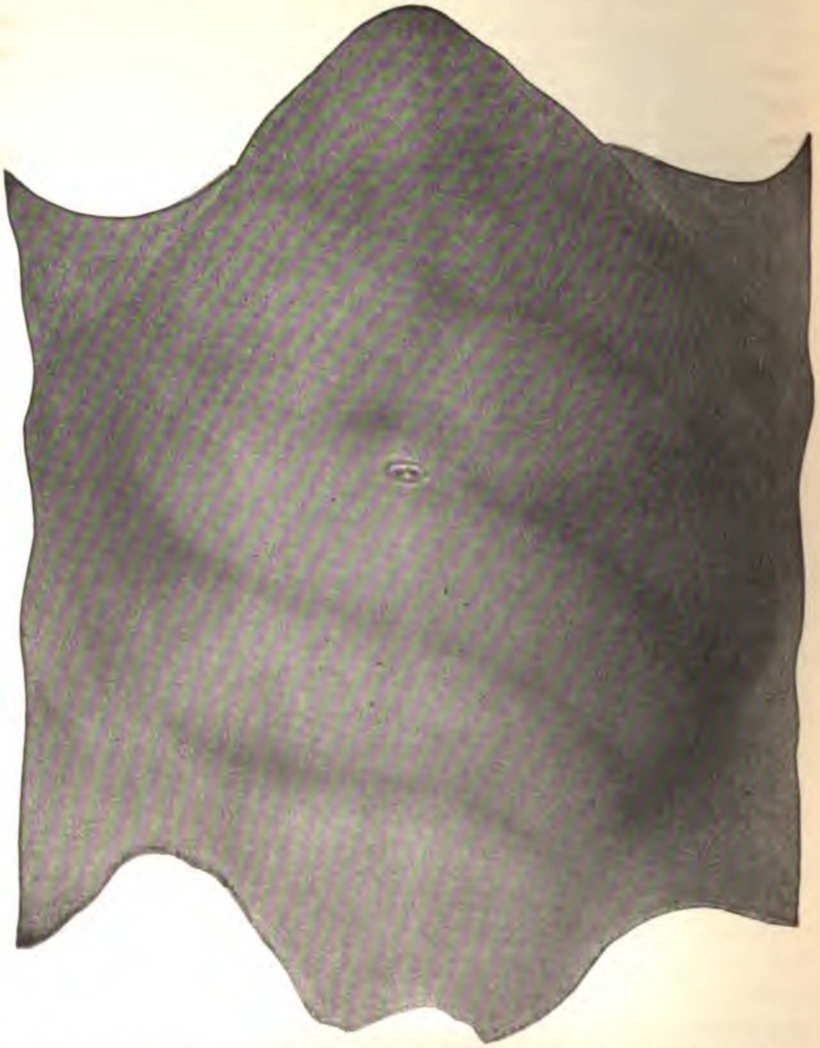


Fig. 200.—Stenosis of the lower ileum from peritoneal adhesion. (Austin after Nothnagel.)

pregnancy. The degree of [illegible] istention is much more considerable in the case [illegible] omen than is the case with primiparæ.

METEORISM

Meteorism or tympanites produces symmetrical abdominal enlargement of transient duration, the abdominal walls being tense, smooth and shiny, affording upon percussion a distinctly tympanitic note, which extends high up and decreases the area of normal dullness of the liver. The umbilicus is level with the adjacent abdominal surface or actually protrudes.

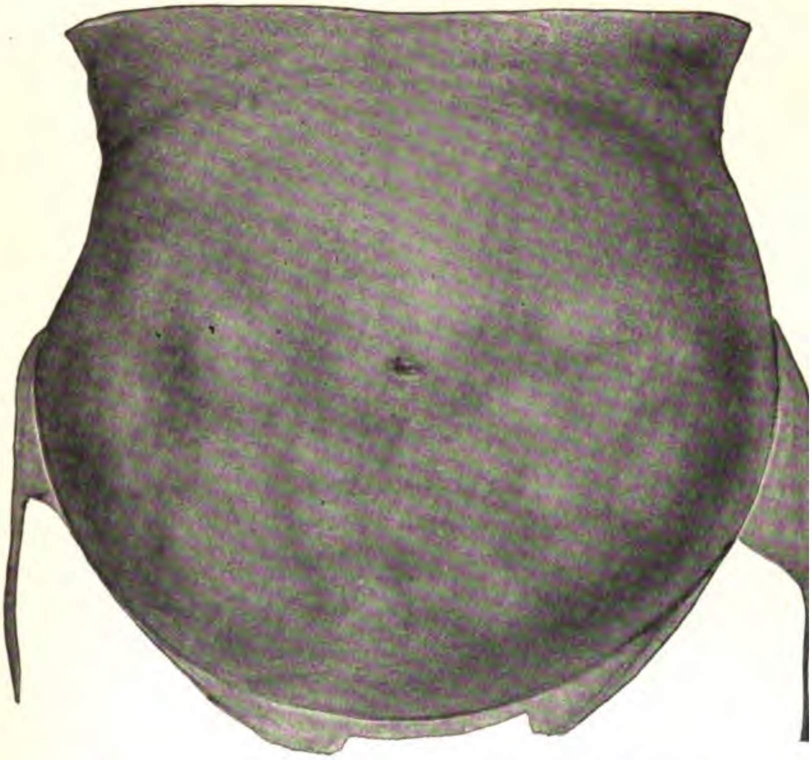


Fig. 201.—Normal intestinal peristalsis. (Austin, after Nothnagel.)

ASCITES

The degree of abdominal enlargement accompanying ascites varies with the amount of fluid in the peritoneal cavity. With the development of the ascitic fluid there is a gradual and uniform enlargement of the abdomen. The contour of the abdomen is characteristically altered, the anteroposterior diameter increasing with moderate flattening of the lateral regions. In the pres-

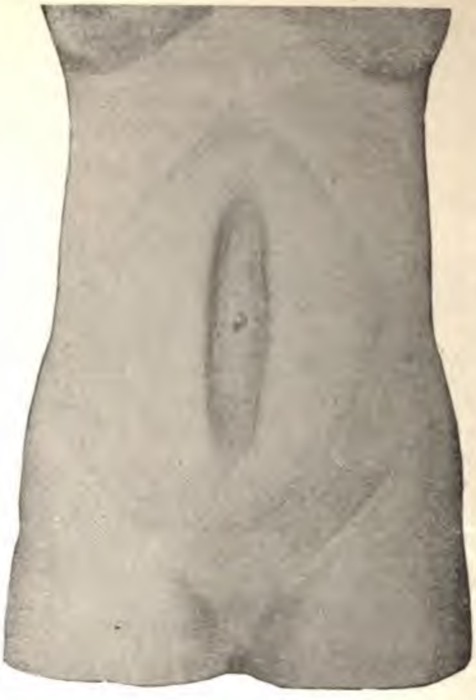


Fig. 202.—Median grooving of the abdominal wall where there is separation of the recti muscles. The woman is represented as lying on her back. (Crossen, after Webster.)

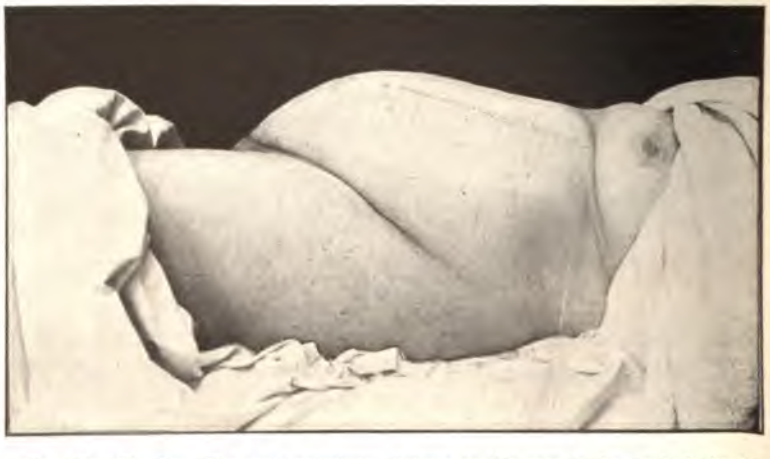


Fig. 203.—Obesity. The most prominent feature in this case is the marked obesity—see Fig. 204. There is also a fibroid tumor of the uterus and a small amount of ascitic fluid. (From Crossen.)

ence of an extensive effusion into the peritoneal cavity the abdominal skin is smooth and tense, and not infrequently it presents silvery striæ. Enlarged, tortuous superficial veins are frequently in evidence.

When the subject of ascites assumes the dorsal decubitus, the percussion note is tympanitic in the median line of the abdomen and is flat in the flanks, owing to the fact that the intestines float upward in the fluid, which gravitates to the dependent portions of the abdominal cavity. When the subject is placed in the lateral decubitus, there is dullness upon the under side and tympany upon the superior side as a result of the same shifting of the intestines with relation to the fluid. Finally, upon placing the subject in the genupectoral position, there is flatness in the umbilical and hypogastric regions, with tympany in the flanks, which are occupied by the resonant intestinal coils.

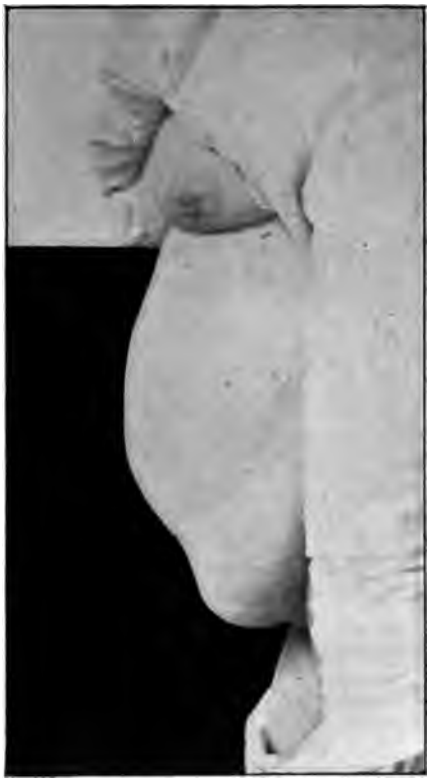


Fig. 204.—Obesity. Patient standing. Same patient as shown in Fig. 203. Notice the thick roll of subcutaneous fat that drops down below the general contour of the abdomen.

VISCEROPTOSIS

Ptosis of the abdominal organs produces characteristic alterations in the abdominal contour with the subject in the erect attitude. Thus, in the presence of gastroptosis, there is an abnormal flattening of the epigastric region with undue prominence of the umbilical region; whereas in the case of enteroptosis, the

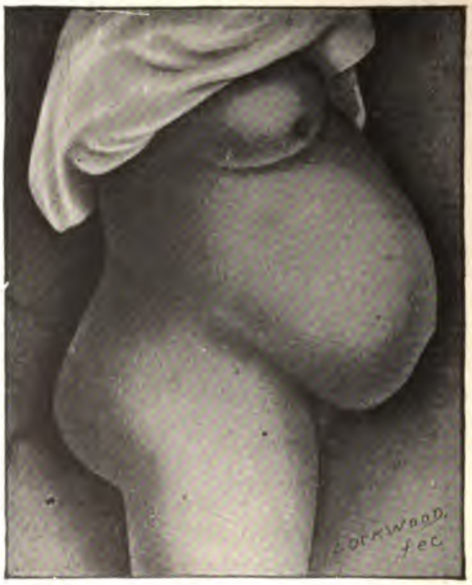


Fig. 205.—Obesity, mistaken for pregnancy by patient. (Crossen, after Williams.)

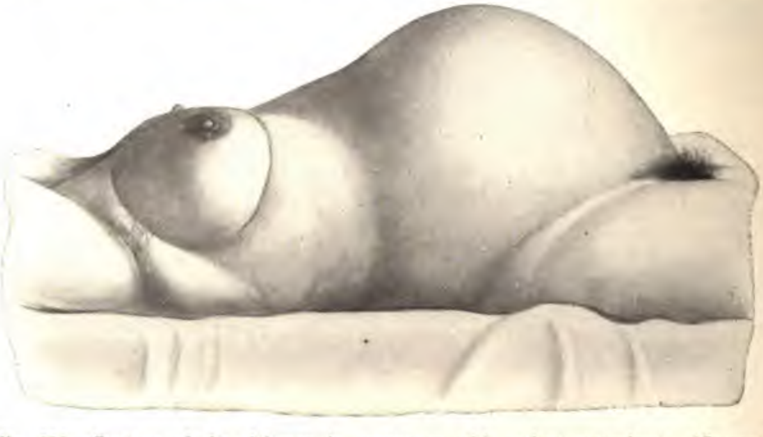


Fig. 206.—Contour of the abdomen in pregnancy with patient recumbent. (Crossen, after Edgar.)

epigastric and umbilical regions of the abdomen are flattened, with marked bulging of the hypogastric and iliac regions, the general contour of the abdomen resembling that of a gourd.

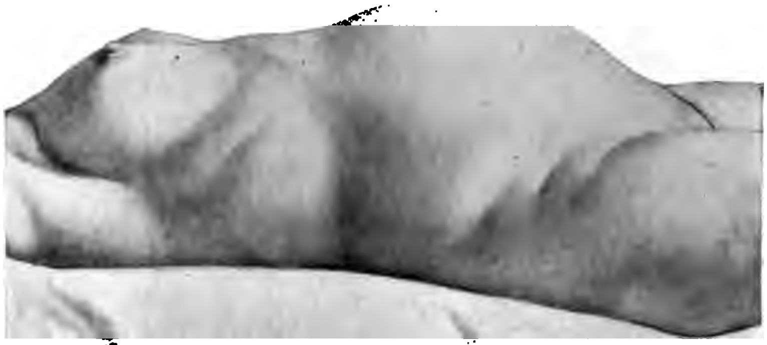


Fig. 207 Tympanites, mistaken for pregnancy by the patient. (Crossen, after Edgar.)

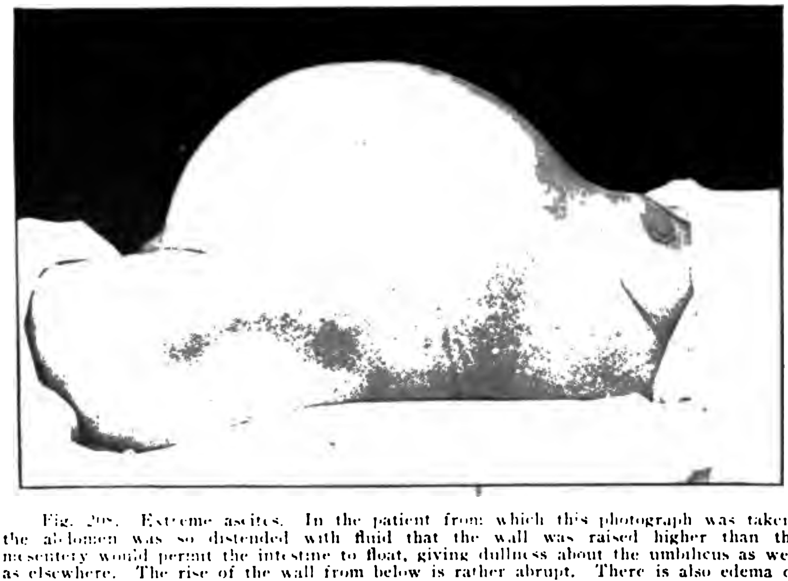
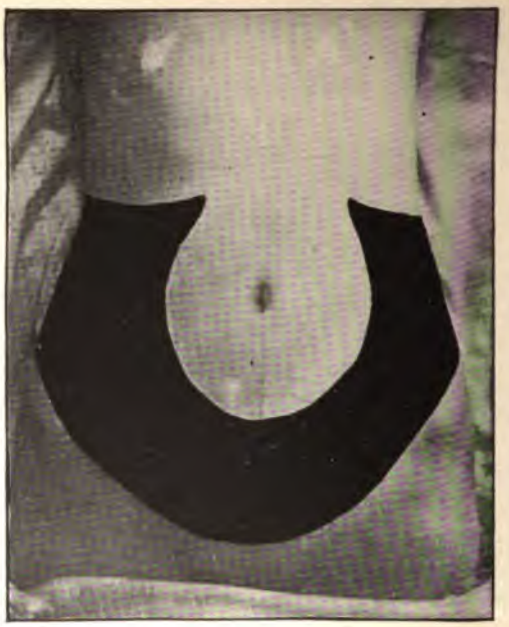
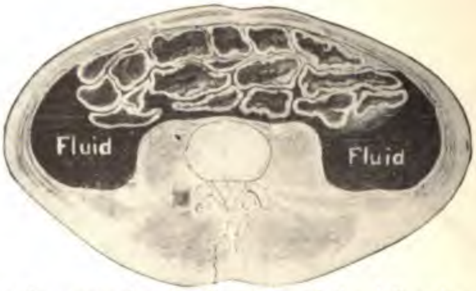


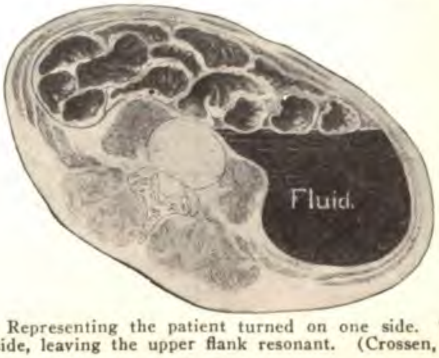
Fig. 208. Extreme ascites. In the patient from which this photograph was taken, the abdomen was so distended with fluid that the wall was raised higher than the mesentery would permit the intestine to float, giving dullness about the umbilicus as well as elsewhere. The rise of the wall from below is rather abrupt. There is also edema of the wall, as shown by the persisting groove where the skirts were tied about the waist. (From Crossen.)



ıowing the area of dullness in moderate ascites, with the patient lying on her back. (From Crossen.)



ıowing the reason for the disposition of the dull and resonant areas in a case of moderate ascites. (Crossen, after Butler.)



scites. Representing the patient turned on one side. The fluid gravitates ınder side, leaving the upper flank resonant. (Crossen, after Butler.)

ASYMMETRICAL VARIATIONS

Local bulging of the epigastric and hypochondriac regions of the abdomen may be significant of distention of the stomach, or enlargement of the liver, gall bladder, or spleen. Bulging of the abdominal wall involving the upper lateral regions of the abdomen and encroaching upon the umbilical region attends enlargements of the kidneys.

Undue prominence of the umbilical region alone occurs with umbilical hernia, and in the case of the progressive enlargement of a neoplasm of the intestine.

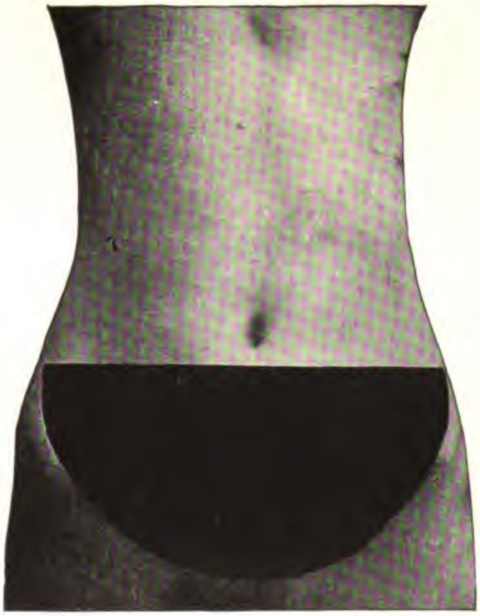


Fig. 212.—Indicating the area of dullness in moderate ascites, with the patient standing. (From Crossen.)

Local bulging of the abdominal wall confined to the hypogastric and iliac regions may be due to a distended urinary bladder, a pregnant uterus, uterine myoma, or ovarian cyst.

The differential points in regard to abdominal enlargement as a result of disease of the various abdominal viscera are discussed in connection with the special examination of the organ in question in subsequent sections.

ABDOMINAL RETRACTION

In the presence of chronic wasting disease, during the course of prolonged diarrhea, and in the presence of stenosis of the cardiac or pyloric orifice of the stomach, the abdominal wall is retracted, the bony landmarks standing out very prominently. The abdomen appears to be "scooped out" like a boat, the scaphoid abdomen.

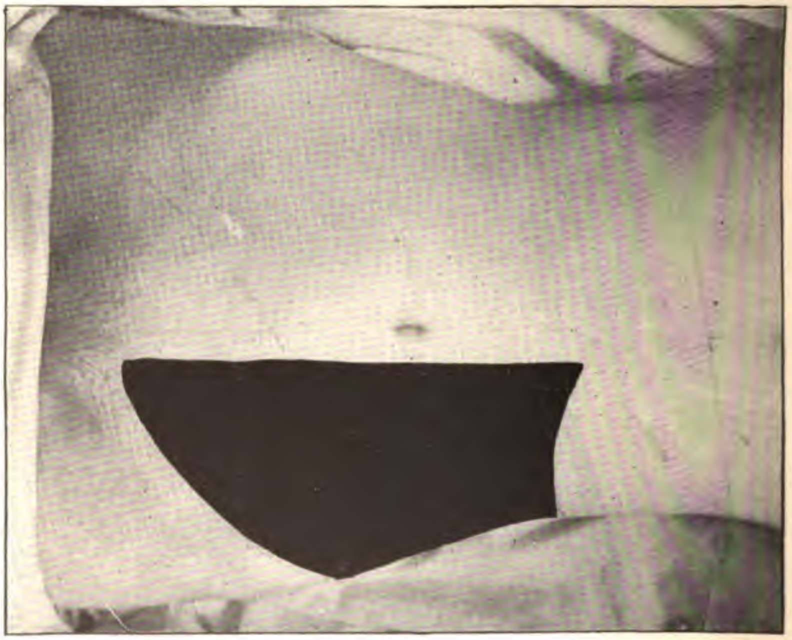


Fig. 213.—Indicating the area of dullness in a case of moderate ascites, with the patient turned on the left side. (From Crossen.)

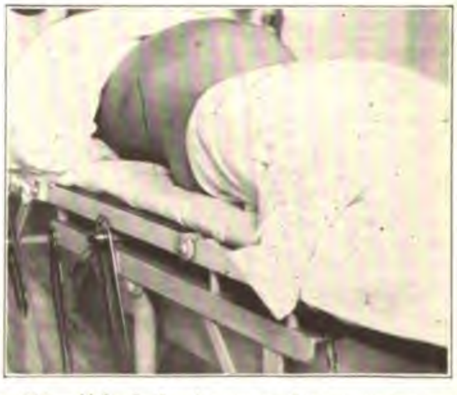


Fig. 214.—Abdominal enlargement due to ovarian cyst.

CHAPTER XXIII

PALPATION, PERCUSSION, AUSCULTATION, AND MENSURATION OF ABDOMEN

PALPATION

Technic.—During palpation of the abdomen the patient should assume the dorsal decubitus with the head slightly elevated by a small pillow and the knees drawn up and supported by a pillow, which relieves the tension of the abdominal wall. In bed-ridden subjects a similar state of abdominal relaxation may be attained by propping the patient's shoulders up with pillows and drawing up the knees and supporting them. Under certain circumstances it is desirable to palpate the abdomen with the patient in the knee-chest position.

The patient having been placed in a natural and unconstrained attitude, he should be directed to refrain from the natural tendency to hold the breath during the examination.

The hands of the examiner should be warm, as a cold hand applied to the surface of the bare abdomen will cause a local muscular rigidity which combats and frustrates the object of the examination. The examining hand should be first applied very gently to the abdominal surface with the palm down and fingers extended, avoiding any sudden pressure or punching movements. During the course of the examination the examiner should first palpate a region which is supposed to be normal before proceeding to the suspected site of disease, as by so doing he gains the confidence and cooperation of the patient.

The abdomen should be palpated systematically, the examiner examining the state of the abdominal wall, palpating any local bulging or retraction which was noted during inspection, and endeavoring to determine whether it is located in the abdominal wall or arises within the abdominal cavity, and the state of the various solid organs of the abdomen, the technic of palpation of which are discussed in their appropriate sections.

The Abdominal Wall.—An estimate of the thickness of the abdominal wall may be made by pinching up the wall between

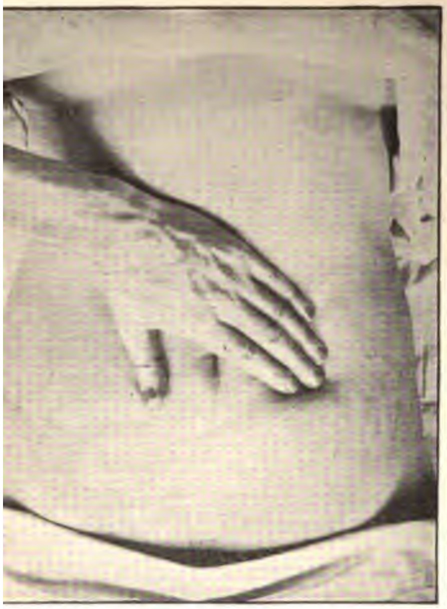


Fig. 215.—Palpation of the abdomen. First p. Hand flat on abdominal surface. (From ssen.)

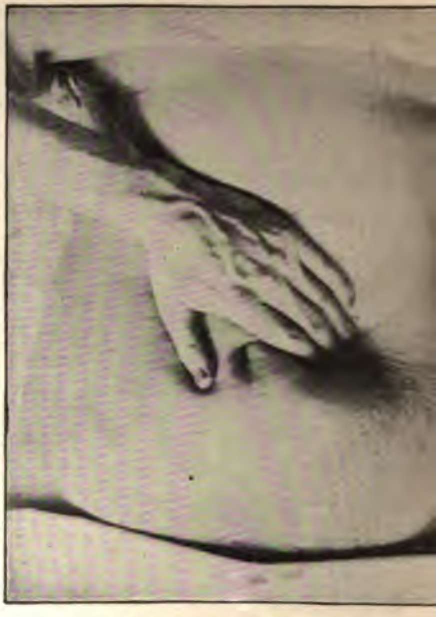


Fig. 216.—Palpation. Depressing the with the fingers of one hand, in various tions. (From Crossen.)

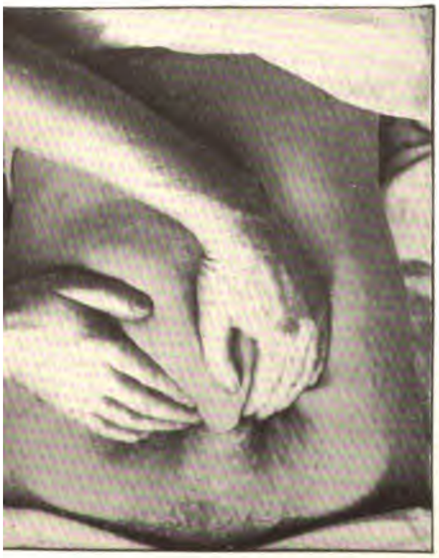


Fig. 217.—Palpation with both hands. (From Crossen.)

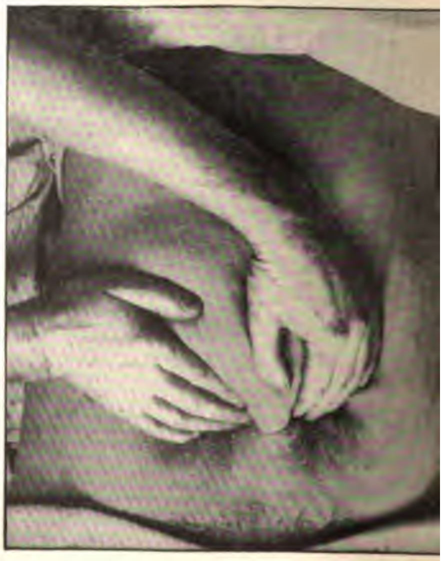


Fig. 218.—Deep palpation with both hands. (From Crossen.)

the forefinger and thumb, or by approximating the two hands placed palm downward upon the abdominal surface. Increased thickness of the abdominal wall indicates an excess of fat, the presence of edema, or suppuration of the wall. If the increase be due to excessive deposition of fat in the wall, a fat wave will be obtained upon bimanual palpation; if due to edema, the wall will pit upon pressure; and if due to a localized or extensive suppuration of the wall, there will be accompanying signs of inflammation, as discoloration of the surface, and elevated temperature.

Rigidity of the abdominal wall with possibly spasm upon attempts at palpation, indicates inflammation of the peritoneum or of an abdominal organ. Muscular rigidity is most commonly encountered in the rectus muscle. Rigidity of the right rectus alone occurs with acute appendicitis, whereas bilateral rigidity of the recti accompanies acute peritonitis.

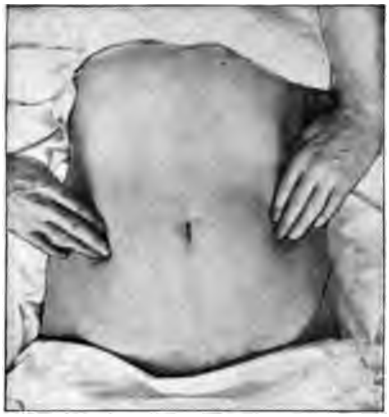


Fig. 219.—Testing the thickness of the abdominal wall. (From Crossen.)

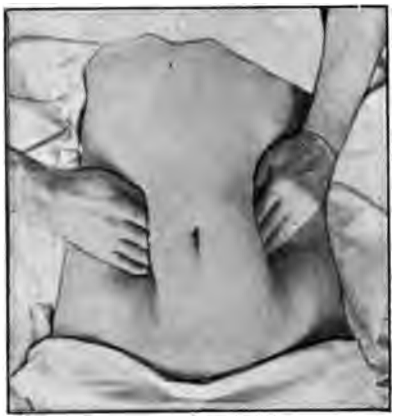


Fig. 220.—Testing the thickness of the abdominal wall.—Second step. The fingers carried beneath the wall. (From Crossen.)

Tenderness.—When tenderness is elicited upon palpation of the surface of the abdomen, if not due to hyperesthesia of the parietes, it points to a diseased abdominal organ. The tenderness is most apt to be encountered over the gall bladder, stomach, spleen, kidney, appendix, and sigmoid flexure. In acute peritonitis there is general or diffuse tenderness.

Fluid Wave.—In the presence of ascites a fluid wave can be demonstrated upon bimanual palpation. In palpating for fluid in the peritoneal cavity, one hand of the examiner is applied flatly over one lumbar region, while the opposite side of the abdomen is tapped with the other hand of the examiner, the finger-tips being used. An impulse or wave is thus created in the fluid, which is appreciable to the palpating hand.

Fat Wave.—Excessive deposition of fat in the abdominal wall gives a wave upon bimanual palpation closely simulating the fluid wave. To exclude such a fat wave during bimanual palpation an assistant should apply the ulnar side of the hand to the median line of the abdomen while the examiner practices bimanual palpation as in eliciting the fluid wave, when the fat wave is inter-

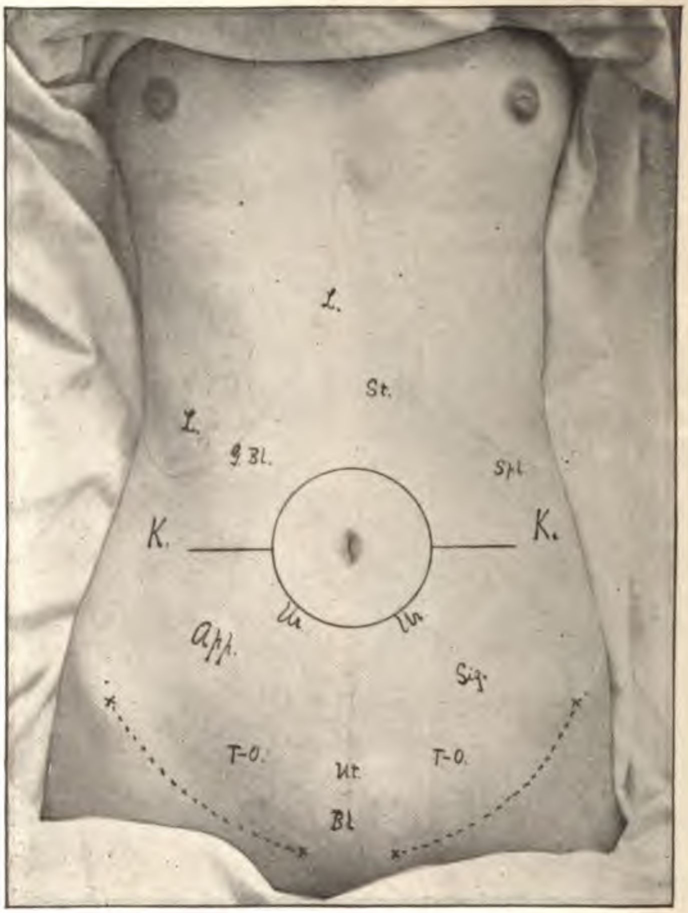


Fig. 221.—Various areas of significant point-tenderness. These are the areas to be investigated during the course of an abdominal examination. (From Crossen.)

rupted by the intervention of the assistant's hand and is not transmitted to the palpating hand of the examiner.

Intraabdominal Tumor.—An intraabdominal tumor may be so large as to entirely fill the abdominal cavity; but as a rule careful bimanual palpation enables the examiner to determine its origin,

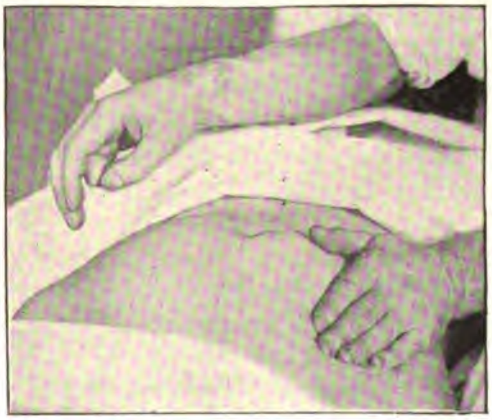


Fig. 222.—Trying for a fluid wave across the abdomen. (From Crossen.)

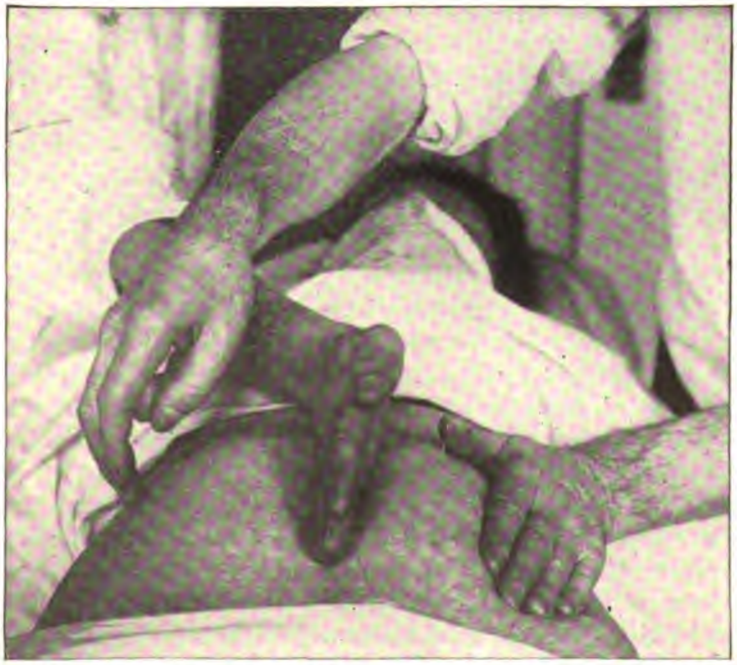


Fig. 223.—Differentiating a fat wave from a fluid wave. The fat wave is stopped by the pressure in the median line. (From Crossen.)

its size and shape, whether it is fixed or movable, and finally whether it moves with respiration. In examining for respiratory mobility of an intraabdominal tumor the examiner should place both hands palms downward flat upon the abdomen, with the fingers directed toward the costal arch, while the patient is directed to breathe deeply. At the commencement of expiration, the finger-tips are pressed downward firmly and with uniform pressure, when the lower margin of the tumor or enlarged organ is encountered. The most commonly encountered movable abdominal tumor is a movable or displaced kidney; but tumors of the liver and spleen are movable with respiration.

Peritoneal Friction Fremitus.—Upon palpating over the upper regions of the abdomen a friction fremitus analagous to pleural or pericardial friction fremitus is sometimes encountered. The vibration is produced by roughening of the peritoneal surfaces in peritonitis. This fremitus is most commonly encountered over the hypochondriac regions, in the presence of peritoneal involvement in the course of perihepatitis or perisplenitis.

PERCUSSION

In the examination of the abdomen the examiner may employ instrumental percussion with the hard rubber pleximeter and the percussion hammer; he may employ ordinary mediate finger percussion; he may resort to flicking percussion; or he may employ auscultatory percussion. In the determination of minor grades of impairment of intestinal or gastric tympany flicking percussion is very serviceable, while in outlining the various solid viscera and tumors of the abdominal organs auscultatory percussion is a very serviceable and dependable method of procedure.

In the practice of flicking percussion, the middle finger of the left hand should be applied upon the abdominal surface with the nail downward, while the middle finger of the right hand is so flexed that the nail of the finger is pressed firmly against the palmar aspect of the thumb. The percussion stroke is delivered by permitting the right middle finger to escape in such manner as to strike firmly and quickly against the palmar aspect of the finger applied to the abdominal surface.

In general, the percussion note is tympanitic upon percussion of the hollow intraabdominal viscera when these contain a moderate quantity of gas unmixed with any excess of solid material. changing to a flat note over the solid intraabdominal organs.

similar flat note is elicited over solid intraabdominal tumors and over collections of fluid, whether encysted or free in the peritoneal cavity.

During percussion of the abdomen the patient should assume the dorsal decubitus with the abdominal wall freely exposed to the pubes. In delimiting the borders of solid organs and tumors, and in the estimation of the relative tympanicity of the adjacent hollow viscera auscultatory percussion, with the employment of a superficial percussion blow or a light stroking movement upon the abdominal surface, is most serviceable. The technic of auscultatory percussion as applied to the examination of the abdomen differs in no wise from the technic which has been described in a previous section.

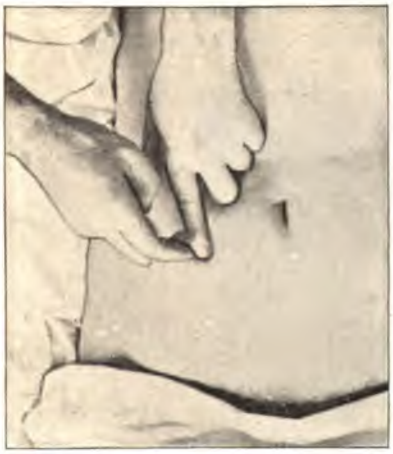


Fig. 224.—Ordinary percussion, which is usually rather superficial. (From Crossen.)

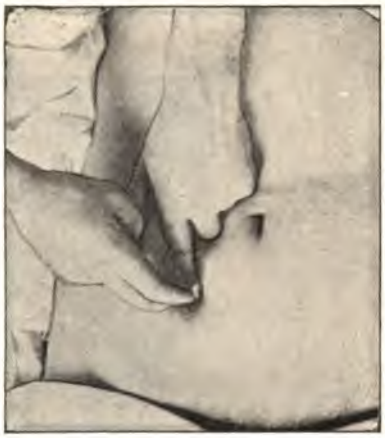


Fig. 225.—Deep percussion. Notice how the left index finger is pressed into the abdomen, so as to thin out the wall and get closer to deep structures. (From Crossen.)

The details of technic of outlining the various abdominal viscera are discussed in their appropriate sections.

AUSCULTATION

Auscultation is seldom employed in the examination of the abdomen and its viscera. Upon auscultation of the abdominal surface overlying the liver or spleen, a friction sound may occasionally be audible in the presence of perihepatitis or of perisplenitis. Similarly, vascular murmurs have occasionally been encountered upon auscultation of the abdomen overlying these organs, as a result of transient constriction of their extensive vascular channels. In cases of aortic aneurysm a vascular murmur may be detected by auscultation over the course of the vessel. In cases of suspected pregnancy, again, auscultation is available in the search for the fetal heart sound as well as for the umbilical or uterine souffle.

MENSURATION

Mensuration of the abdomen is practiced in order to determine variations in the contour and extent of the abdominal walls in the presence of intraabdominal tumors, tympanites, ptosis, or cysts. Successive mensuration is practiced to determine any progressive enlargement of the abdomen from these conditions as well as to arrive at conclusions as to any diminution in these dimensions.

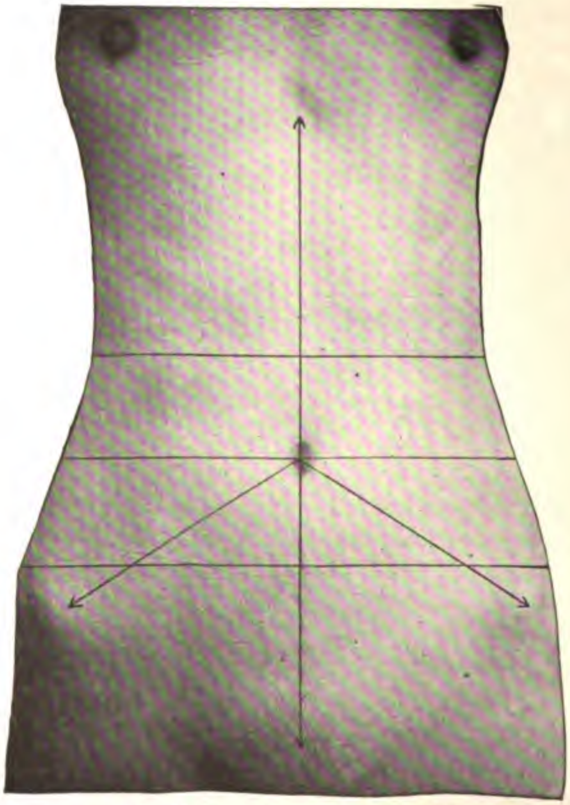


Fig. 226.—Showing the lines for mensuration. (From Crossen.)

In the practice of mensuration of the abdomen seven lines are employed; namely, (1) a line encircling the trunk at the level of the umbilicus; (2) a line encircling the trunk three inches above the umbilicus; (3) a line encircling the trunk three inches below the umbilicus; (4) a line extending from the umbilicus to the right anterior superior iliac spine; (5) a line extending from the umbilicus to the left anterior superior iliac spine; (6) a vertical line extending from the umbilicus to the tip of the ensiform process; and (7) a vertical line projected from the umbilicus to the symphysis pubis.

SECTION II

SPECIAL EXAMINATION OF THE ABDOMINAL VISCERA

CHAPTER XXIV

THE STOMACH, INTESTINES, AND PANCREAS

EXAMINATION OF THE STOMACH

Clinical Anatomy.—The stomach occupies the epigastric and left hypochondriac regions of the abdomen when the organ is normal. The *cardiac orifice* of the stomach is located behind the seventh left costal cartilage, at a point one inch from the sternum. The position of the *pyloric orifice* is somewhat variable, its site being modified by the condition of the stomach. When the stomach is empty, this orifice occupies a position in the median line of the abdomen, at a point midway between the episternal notch and the symphysis pubis; when the stomach is moderately distended, the pylorus occupies a position approximately one inch to the right of the median line at the same level, this displacement to the right being increased to two or three inches in the presence of extreme distention of the organ.

The *fundus* of the stomach is in contact with the inferior aspect of the diaphragm, behind and below the apex of the heart, in which position it extends as high as the sixth rib. The *lesser curvature*, representing the superior limit of the stomach, is covered by the left lobe of the liver, passing downward and toward the right from the cardiac orifice to the pyloric orifice of the stomach. The *greater curvature*, representing the inferior limit of the organ, crosses the left costal arch at the level of the ninth left costal cartilage, the most dependent portion of the normal organ occupying a level approximately two inches above the umbilicus.

The *anterior surface* of the stomach, largely overlapped
left lobe of the liver, inferior border of the left lung
costal arch, is exposed to the anterior
limited portion of its
in which the anterior
the anterior abd

gastric tympany, is limited superiorly and upon the left by the left lobe of the liver, superiorly and upon the right by the inferior border of the left lung, and inferiorly by the transverse colon.

The *posterior surface* of the stomach is directed downward and backward, reposing in the so-called "stomach bed," formed by the transverse mesocolon, the pancreas, left kidney, and suprarenal capsule.

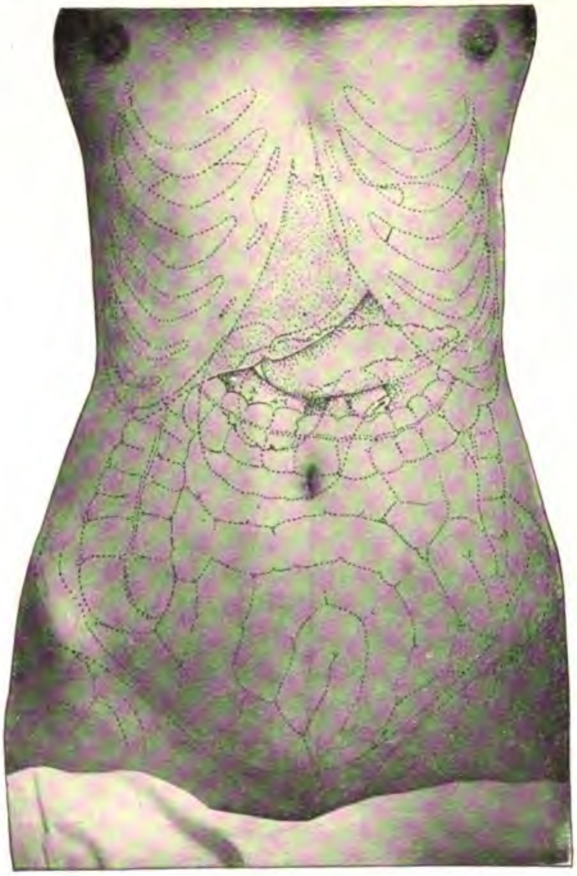


Fig. 227.—The central upper abdomen. Showing in outline the liver and stomach and pancreas. (From Crossen.)

Physical Examination.—*Inspection.*—In practicing inspection of the abdomen for the detection of affections of the stomach, the patient should in the first place assume the dorsal decubitus, with a free exposure of the abdomen, the thorax, and the lower cervical region. With the subject in the recumbent posture, the examiner should assume a position with his eyes upon the level of the abdominal surface, and should inspect this surface from the left side, from the right side, from the feet of the patient, and from the head of the patient, with oblique illumination of the abdominal and thoracic surfaces.

The normal stomach in the subject with abdominal walls of normal thickness gives no evidence of its presence upon inspection of the epigastric and left hypochondriac regions. But, in the presence of diastasis of the rectus muscles, and in the emaciated subject who has lost much of the paniculus adiposus of the abdominal wall, if the stomach is at all distended with gas, it is possible to observe a protrusion of the epigastric region, to which is not infrequently added a bulging of the left costal arch. In the presence of diastasis of the recti, visible peristalsis is frequently noted, the peristaltic waves being recognized as a vermicular movement upon the abdominal wall which travels from the left side of the epigastrium toward the right and downward. Bamberger has described visible peristalsis in connection with pyloric stenosis in which, following a visible constriction of the central portion of the stomach, the peristaltic waves were observed to pass in opposite directions toward the cardia and toward the pylorus.

When epigastric bulging of gastric origin is observed upon inspection of the epigastrium, the first point to be settled is the site of the greater curvature of the organ, representing the inferior limit of the stomach; and the second detail is to determine whether the lesser curvature has departed from its normal site. In the presence of gastric dilatation and gastroptosis the greater curvature is frequently encountered below the level of the umbilicus, and it may be as low as the pubes. Its lower limit when the organ is distended with gas is indicated by a transverse elevation with its convexity directed downward. It is to be borne in mind, however, that epigastric bulging is usually due to gaseous distention of the stomach, and that pronounced gastrectasis may exist without this bulging, provided that the stomach is not filled with gas. In fact, in true gastrectasis epigastric bulging is frequently absent, and the abdominal wall is frequently scaphoid and flaccid. Hence, gastrectasis and gastroptosis are by no means excluded by the absence of epigastric bulging, and further exploratory methods must be employed to determine their presence or absence in any case.

In suspected cases of gastrectasis or gastroptosis, after the patient has been examined in the recumbent posture, a further inspection of the abdomen should be conducted with the subject in the standing posture with a view to the detection of the more or less characteristic changes in the contour of the abdomen in these affections.

The determination of the level of the lesser curvature of the stomach is extremely important in dealing with suspected cases of gastrectasis and gastroptosis; but, as this curvature is covered by the left lobe of the liver, it is inaccessible to inspection in the normal subject; and even in the presence of gastroptosis, it is ordinarily necessary to resort to artificial distention of the stomach to render it accessible to inspection.

The artificial distention of the stomach after the method of Frerichs is very serviceable in this connection. The subject is given a dram of sodium bicarbonate in solution, which is shortly followed by a dram of tartaric acid in solution. The reaction of the two solutions generates carbon dioxide in the stomach, which serves to distend the organ. Frequently this artificial distention of the stomach is attended by mild symptoms, such as transient dyspnea, anxiety, and acceleration of the pulse, as a result of increased subphrenic pressure exerting pressure upon the heart. These symptoms are, as a rule, transient, and are entirely relieved by a few eructations of gas. If they persist and are distressing to the patient, they are immediately relieved upon the passage of a stomach tube, with evacuation of the gas.

Artificial distention of the stomach has been practiced by forcing air into the viscus through a stomach tube by means of a rubber bulb. In this procedure it not infrequently happens that there is produced a simultaneous distention of the entire intestinal tract, with very little distention of the stomach. The pyloric orifice does not seem to react to the inflation of atmospheric air and to close as it does in the presence of carbon dioxide. Moreover, the inflation with atmospheric air necessitates the employment of the stomach tube, which is annoying to the subject of the examination.

In the presence of gastroptosis and gastrectasis in a patient with abdominal walls which are not loaded with fat, upon artificial distention of the stomach the two curvatures of the viscus may be detected upon inspection, as the stomach assumes a more intimate contact with the abdominal wall. In the presence of gastrectasis it is further observed that the lesser curvature retains its normal position, while the greater curvature extends abnormally low in the abdominal cavity, occasionally below the umbilicus or even to the pubes.

In certain cases of pyloric tumor inflation of the stomach causes a knotty protuberance to become visible in the region of the pylorus. Most frequently these tumors are located to the right and a little above the level of the umbilicus. It is noted in the case of gastric tumors that they do not move with the respirations, a fact which serves to distinguish them from tumors of the liver or of the spleen, which possess respiratory mobility. It is possible, however, for a gastric tumor to exhibit respiratory mobility if adhesions have become established between the new growth and the liver, in which event the movements of the liver are communicated to the gastric tumor. Occasionally a gastric tumor presents a systolic elevation as a result of an impulse which is communicated to the tumor from the subjacent abdominal aorta. In this event, upon placing the patient in the genupectoral position the tumor falls away from the aorta and the systolic elevation is obviated.

Einhorn, Kuttner, Jacobson, and others have introduced various types of illuminating apparatus into the cavity of the stomach with the view of studying the gastric outlines by the medium of transmitted light, a method of examination which was designated by Einhorn, *gastrodiaphany*. Up to the present time gastrodiaphany has not been demonstrated to possess greater clinical value than the simpler methods of exploration, as transillumination of adjacent viscera is quite possible during the procedure, with consequent erroneous conclusions.

Palpation.—In practicing palpation of the stomach, the patient should assume the dorsal decubitus with the head comfortably elevated by a pillow and the legs drawn up and properly supported, in order to relieve the muscles of the abdominal wall from undue tension. The examiner, seated upon the left side of the patient, should apply the palms of the hands flat upon the epigastrium immediately below the tip of the ensiform cartilage and gradually sink them into the abdominal wall, covering this area progressively in a direction from above downward. Following this maneuver, he should apply the tips of the fingers of the right hand to the lower portion of the epigastrium; and, with the fingers well separated, search for the greater curvature of the stomach by a series of gentle, pushing movements.

Palpation of the epigastrium may reveal the presence of tenderness, may confirm visible peristalsis, or the presence of a tumor of the stomach, or may elicit succussion sounds.

Tenderness elicited in the epigastric region, in which such a large number of vital structures lie within a limited space, natural possesses a varied significance. It may be signifi inflammation of the mucous lining of the stomach, of ulcer or carcinoma of the stomach, of pancreatic carcinoma or carcinoma of the common bile duct, or of acute hepatitis, hepatic colic, pancreatic calculus, or acute pancreatitis.

In the case of gastric ulcer the tenderness is circumscribed in the majority of cases to a point corresponding to the junction of a vertical line drawn from the umbilicus to the tip of the ensiform cartilage, and of a horizontal line connecting the free margins of the eighth costal cartilages. Moreover, gastric ulcer is commonly attended by a painful pressure point situated immediately to the left of the body of the twelfth dorsal vertebra.

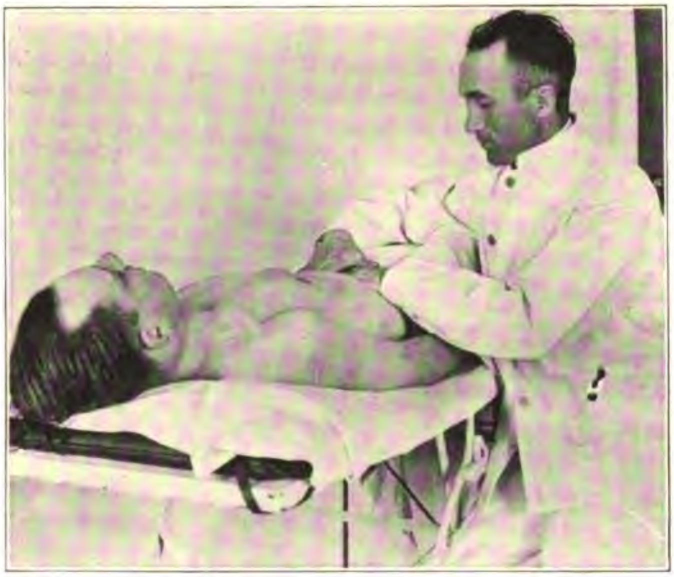


Fig. 228.—Palpation of the epigastrium.

The tenderness of acute gastritis and of gastric carcinoma are diffuse, pervading all portions of the epigastrium upon deep pressure. Occasionally, also, in the case of gastric carcinoma it is possible, in patients with thin abdominal walls or with diastasis of the recti, to palpate the carcinomatous nodules upon the anterior gastric wall.

A solid tumor of the stomach may occupy the anterior wall, the posterior wall, or the pylorus. A pyloric tumor is commonly palpable in a circumscribed area to the right of the umbilicus and a little above this level. Moreover, pyloric tumors are commonly attended by varying grades of pyloric stenosis with visible peristalsi

and an abnormally low position of the greater curvature
stomach. The stomach in this state commonly yields distir
cussion sounds even several hours after the ingestion of f
fluid.

A solid tumor of the anterior gastric wall is readily p
by ordinary methods; but in the uninflated stomach it is imp
to say whether a palpable tumor is situated upon the anterior
posterior wall of the viscus. In the determination of the
the tumor under these circumstances artificial inflation
stomach with carbon dioxide is very serviceable. When
tended, the tumor situated upon the posterior wall is no
freely palpable.

The soft, yielding walls of the normal stomach are not pa
except in the cases in which the organ has been artificially in
In the artificially inflated stomach the examiner feels upon
tion of the epigastrium a sensation of resistance when the st
is reached, a sensation closely akin to that which is experi
upon palpating a rubber bag filled with gas. In the prese
gastrectasis and gastroptosis the greater curvature of the
is encountered at an abnormally low level; and in the ca
gastroptosis it is frequently possible in the inflated orga
palpation to outline the lesser curvature of the organ and
tablish the fact that the superior limit of the stomach has dep
from its normal habitat beneath the left lobe of the liver.

Succussion sounds arising within the stomach are elicite
palpating the epigastrium with a series of short pushing
ments with the finger-tips, beginning well below the normal s
the greater curvature and proceeding upward until this poi
reached whereupon the splashing sounds will become audibl
is not sufficient, however, to cease the palpation when the so
become audible, unless at the same time the fluid becomes palp

Gastric splashing sounds were first described by Chomel,
regarded them as an invaluable sign of gastrectasis; and so the
if they fulfill certain topographical and chronological condit
These conditions have been established by Bouchard; namely,
splashing sounds are indicative of gastrectasis when they ex
well beyond the limits occupied by the normal organ; and
they are elicited at least six hours after the ingestion of foo
fluid.

Glenard's Belt Sign.—In the presence of gastroptosis,
the examiner, standing behind the patient, places his hands
the lower portion of the abdomen and lifts upward and backw

the patient with gastroptosis experiences a sensation of relief from the dragging sensation which accompanies this disease.

Percussion.—Percussion of the stomach is employed to determine the size, shape, and position of the viscus. During the examination as in the case of palpation and inspection of the epigastrium, the subject should occupy the dorsal decubitus. In the examination of the organ by percussion, finger percussion, percussion with the hammer, or flicking percussion may be employed with uniformly good results in the course of the examination.

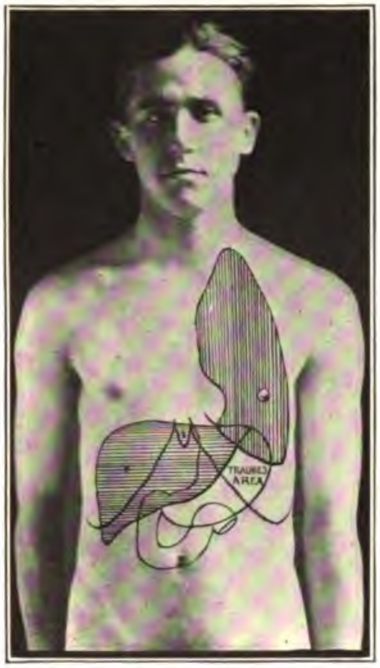


Fig. 229-*A*.—Traube's semilunar space.

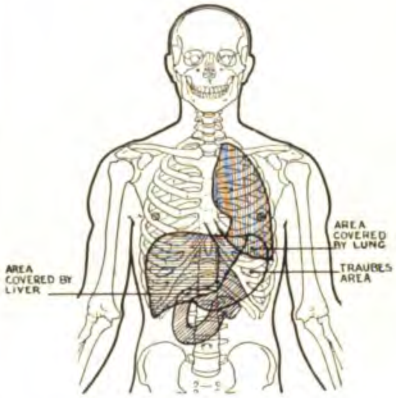


Fig. 229-*B*.—Traube's semilunar space.

The findings upon percussion of the stomach vary with the state of the organ. The sound which is elicited depends upon the contents of the stomach and upon the tension of its walls. According to the different variations in these factors the gastric cavity is capable of generating pure tympany, a metallic sound, or frank flatness. As the stomach is continually presenting active contractions and relaxations, the percussion sound which is elicited naturally changes with great frequency during a brief period of time.

Pure gastric tympany is only obtained in Traube's semilunar space, when the stomach is moderately distended and its walls ar not under undue tension. This area, corresponding to the portion of the anterior gastric wall which is in direct apposition with the thoracic and abdominal surface, presents a transverse diameter of four to five inches and a vertical diameter of three to four inches. The space possesses three distinct limits which may be determined by alterations in the quality and pitch of the percussion sound. Superiorly and to the left the gastric tympany is interrupted by the inferior border of the left lung, the *gastropulmonary limit* of the space; superiorly and toward the right it is interrupted by the left lobe of the liver, the *gastrohepatic limit;* while inferiorly it is interrupted by the colonic tympany of the transverse colon, constituting in this situation the *gastrocolic limit* of gastric tympany. In the minimal number of cases in which the tip of the left lobe of the liver does not reach the apex of the heart, there exists yet another change in the percussion sound in this situation, constituting a *gastrocardiac limit* of Traube's space.

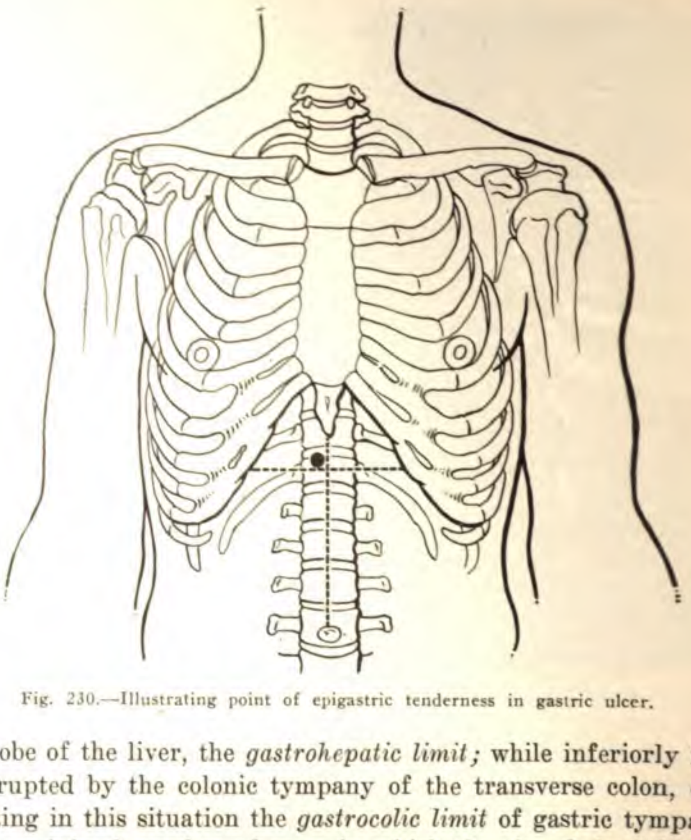


Fig. 230.—Illustrating point of epigastric tenderness in gastric ulcer.

The gastropulmonary and gastrohepatic boundaries of the semilunar space of Traube are readily determined by mediate per sion with the fingers from an area of frank gastric tympany in central portion of this area toward the lung and the liver respectively. The delineation of the gastrocolic boundary of the space is more difficult, owing to the fact that it is possible for the gastric and the colonic tympany to approximate one another in quality and pitch. This difficulty is obviated by the introduction of a liter of fluid into the stomach prior to the examination. Upon percussing upward under these conditions, the colonic tympany gives place to dullness or flatness, when the greater curvature of the stomach is reached.

Auscultatory percussion is to be preferred, however, in the accurate determination of the size, shape, and position of the stomach. In this method of examination the chestpiece of the stethoscope is applied at the center of gastric tympany in Traube's space. The examiner delivers a few blows and fixes in his mind the quality and pitch of the sound elicited. Without moving the position of the bell of the instrument, he then begins at several points upon the abdominal surface and percusses toward the bell of the stethoscope. In each instance a change in the quality of the percussion note will indicate when the gastric border has been reached.

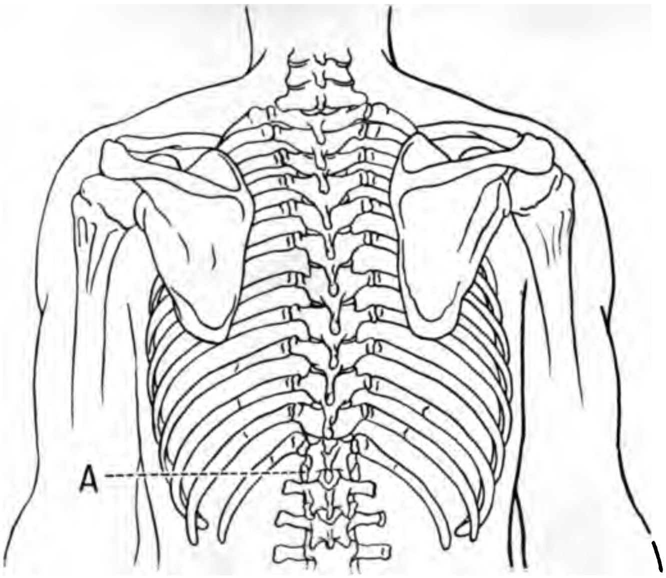


Fig. 231.—Illustrating dorsal pressure point in gastric ulcer.

Scratching percussion is also very serviceable in delimiting the borders of the stomach. In this procedure the examiner applies the bell of the stethoscope to the area of frank gastric tympany in Traube's semilunar space and scratches the skin of the abdomen in this area with the nail. Having fixed in his mind the quality and pitch of the sound elicited, he begins at various regions of the abdominal surface and passes the finger-nail along the skin toward the bell of the instrument. A change in the quality of the sound is noted in each instance when the gastric borders are reached. The rubber tip of the percussion hammer is very serviceable in scratching percussion, as is also the eraser upon the end of a lead pencil, which is drawn across the skin toward the bell of the instrument. In each instance the quality of the sound changes abruptly when the gastric borders are attained.

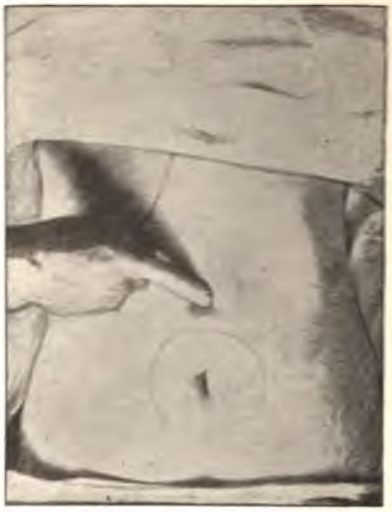


Fig. 232.—Showing the region for tenderness or a mass from disease of the stomach or pancreas. (From Crossen.)

An *extension* of the area of gastric tympany does not in every case in which it is encountered indicate an increase in the volume of the organ. In the presence of atrophic changes in the liver the left lobe of this organ is retracted toward the median line and the gastric tympany is extended in this direction, with the consequent production of a gastrocardiac limit of the space of Traube. Similarly, in the presence of retraction of the anterior and inferior borders of the left lung in the course of chronic interstitial pneumonia, fibroid phthisis, or pulmonary syphilis, the gastric tympany is extended in a direction upward and outward as a result of retraction of the gastropulmonary limit of this space.

Extension of the area of gastric tympany as a result of enlarg ment of the organ itself is encountered in the presence of gastrectasis and in congenital enlargement of the organ, (megalogastria). In gastroptosis, on the contrary, although the inferior limit of the area of gastric tympany is markedly lowered, it can be determined by careful percussion that the lesser curvature no longer resides in its normal habitat beneath the left lobe of the liver. Again, in the case of gastrectasis, an error is apt to arise through the accumulation of fluid in the dilated stomach if care is not exercised to evacuate this fluid prior to the examination.

Diminution of the extent of gastric tympany is by no means invariably associated with diminution in the size of the stomach. In hepatic enlargements the left lobe of the liver encroaches upon the semilunar space of Traube and causes downward displacement of the gastrohepatic limit of this space. Similarly, in the presence of left-sided pleural effusions and pyopneumothorax, the gastropulmonary limit of the space is lowered with consequent diminution in the limits of gastric tympany. Pitres has noted diminution in the dimensions of gastric tympany in connection with extensive effusion into the right pleural sac, which he attributes to crowding over of the liver by the weight of the effusion. The presence of varying quantities of solids and fluids in the stomach diminishes the area of gastric tympany.

Diminution of the area of gastric tympany which is referable directly to diminution in the size of the stomach is occasionally demonstrable in the presence of cardiac stenosis from malignant disease of the cardiac orifice of the stomach. However, in this event the atrophic stomach as a rule is pushed upward and backward by the transverse colon and in no wise enters into the tympany which is produced upon percussion in Traube's semilunar space. Indeed, Dehio has demonstrated, both upon the living subject and upon the cadaver, that when the normal stomach is empty the tympany which is obtained upon percussion of Traube's space is invariably due to the transverse colon, which crowds the empty stomach upward into the left concavity of the diaphragm.

An hour-glass constriction of the stomach may be demonstrated by the introduction of water into the stomach; when, upon percussion over the viscus, it is observed that the stomach is not uniformly distended; but that the cardiac portion is distended, while the pyloric portion remains empty; and, moreover, that in a short time fluid passes into the pyloric portion, which in turn becomes distended. If, during this time, the stethoscope be applied over the central portion of the stomach, it may be possible to hear the water gurgle through the constricted portion of the organ.

Auscultation.—Succussion sounds which are generated in the stomach and which are audible during palpation possess a correspondingly greater intensity upon auscultation of the epigastrium, and are propagated to a considerable distance from their site of production. In eliciting these sounds by auscultatory percussion, the patient should assume the dorsal decubitus with the abdominal muscles relaxed. The bell of the stethoscope is placed over the central portion of Traube's space and the epigastrium is gently tapped with the finger-tips of the opposite hand of the examiner, whereupon the examining ear appreciates a series of sounds analogous to those which are produced upon shaking a rubber bag which is partially filled with fluid. Demonstrable in the normal stomach when it contains coincidentally air and fluid, the succussion sound possesses a much greater range of intensity and area of propagation in the case of gastrectasis.

Upon auscultation of the stomach in the presence of gastrectasis with active fermentation of the gastric contents, the examiner frequently encounters a series of fine crackling sounds, caused by the bursting of minute bubbles upon the surface of the contained fluid, the enlarged stomach amplifying and acting as a resonator for the sounds. These sounds are never audible below the inferior curvature of the stomach; and, when they are demonstrable in the presence of a dilated stomach, serve as a reliable guide in checking the findings upon palpation and percussion of the organ.

Kronicker and Meltzer first called attention to two deglutition sounds which are audible upon auscultation of the superior portion of the epigastrium immediately below the tip of the ensiform cartilage. The first of these sounds is simultaneous with the act of deglutition, while the second which is attributed to the passage of the ingested fluid through the cardiac orifice, is audible approximately seven seconds after the first sound. In the presence of partial stenosis of the cardiac orifice of the stomach the second sound is replaced by an irregular spouting, churning sound as the ingested fluid passes through the constricted orifice. In the presence of complete stenosis of the cardia, the second deglutition sound is abolished.

EXAMINATION OF THE SMALL INTESTINE

Clinical Anatomy.—The small intestine, the section of tl gastrointestinal tract extending from the pyloric orifice of stomach to the junction with the large intestine at the ileocecal valve, lies within the frame formed by the course of the large intestine, slightly overlapping the ascending and the descending colon, and extending for a variable distance below the brim of the pelvis.

The *duodenum,* the proximal twelve inches of the small intestine, pursues a course resembling the letter "C" from the pylorus to the duodenojejunal flexure at the left side of the second lumbar vertebra, embracing in its course the head of the pancreas and the common bile duct, which empties its contents into this portion of the small intestine.

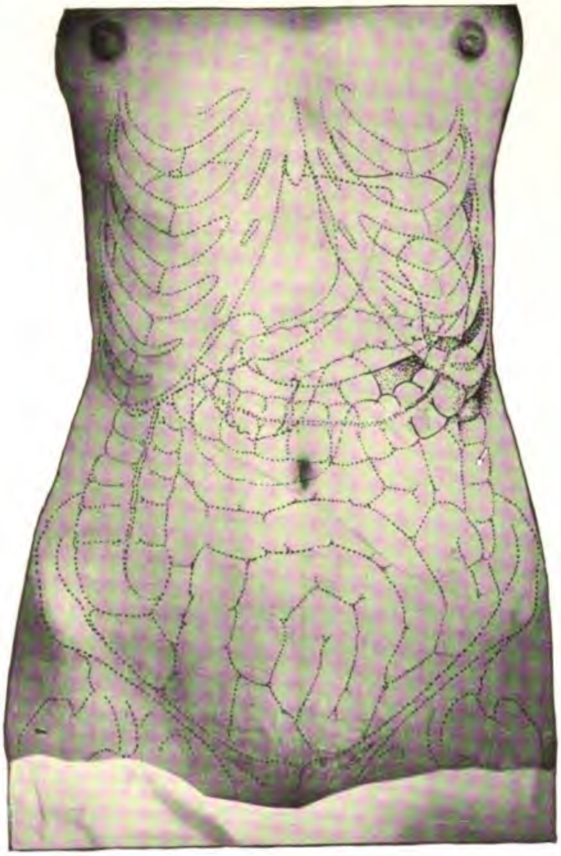


Fig. 233.—The left upper abdomen. The site of the spleen and of the splenic flexure of the colon, the organs in this region most commonly affected, are shown by the stippling. When normal, the spleen lies considerably higher in the abdominal cavity than is generally supposed. Its anterior projection is shown here in dotted outline, with the lower end in contact with the splenic flexure of the colon. (From Crossen.)

The *jejunum*, the second division of the smal
prising approximately eight feet of the tube, lies
and right and left lumbar regions, and is freely m

The *ileum*, the distal twelve feet of the small in
terminates at the ileocecal valve, lies in the umbilical,
and lumbar regions, in which it is freely movable. Or
omentum intervenes between the jejunum and ileum ar
rior abdominal wall.

Physical Examination.—*Inspection.*—In the norma
with well-developed abdominal walls inspection of the
yields no evidence of the presence or location of the coil
small intestine. In the emaciated subject, however, and in
presenting diastasis of the rectus muscles, visible perista
occasionally detected in the absence of intestinal pathology; a
the presence of intestinal obstruction this peristalsis is gr
exaggerated. If the site of the obstruction is in the lower por
of the ilium, near the ileocecal valve, the peculiar "ladder
tern" is frequently noted, occupying the umbilical region.

Palpation.—The soft yielding walls of the normal small intestin
are not to be felt upon palpation of the abdominal wall. But in
the subject with thin abdominal walls enteroliths or large gall-
stones may occasionally be detected by palpation if they are pres-
ent. A hard tumor which is encountered in the neighborhood of
the umbilicus and in the lower umbilical and upper hypogastric
regions is very apt to belong to the small intestine, and may be
significant of malignant disease, of intussusception, or of volvulus.
A solid mass encountered in these regions, however, is occasionally
due to matting of the omentum in tuberculous peritonitis, consti-
tuting tabes mesenterica.

When an intestinal tumor pulsates, the patient should be placed
in the genupectoral position, in order to determine whether the
ulsation is transmitted to the tumor from the subjacent abdominal
rta. When palpation of the intestine is practiced in this posture,
tumor falls away from the aorta, and the pulsation of this
el is no longer imparted to the growth.

rcussion.—The note which is elicited upon percussion of the
intestine varies in pitch and quality with the amount of
ned in the tube and with the tension of its walls. The norma
e yields a tympanitic or metallic percussion sound, but
sence of a considerable content of solid material, the
dull. When the intestine is greatly distended with
obstruction, the note is frankly tympanitic, but of

lower pitch than in the case of percussion of the normal intestine with only moderate tension of its walls. A similar percussion note is encountered in the presence of a solid tumor which is surrounded by tympanitic intestine. The same note is encountered in the presence of multiple incomplete stenoses of the intestine as the result of tuberculous ulceration. Frequently under these circumstances there is accumulation of fluid above the level of each of the obstructions, yielding impairment of intestinal tympany in multiple areas of the abdominal surface, constituting the sloping dullness of Mathieu and Ricard.

Auscultation.—Upon auscultation of the abdominal wall over the distribution of the small intestine, the examiner frequently encounters transient gurgling sounds, *borborygmi,* which are without diagnostic significance. But in the presence of a partial obstruction of the intestine, auscultation at the level of the stenosis reveals a more intense fluid sound, resembling that which is produced by the sudden expulsion of liquid from a syringe. In the presence of multiple partial intestinal stenoses due to tuberculous ulceration König has encountered these fluid sounds at varying levels of the abdominal wall, accompanied by succussion sounds upon palpatory percussion and by the sloping dullness of Mathieu and Ricard.

EXAMINATION OF THE LARGE INTESTINE

Clinical Anatomy.—The large intestine, comprising the terminal six feet of the gastrointestinal canal, comprises the cecum with the vermiform appendix, the ascending, transverse, and descending colon, the sigmoid flexure and the rectum. The large intestine is arranged in the form of a frame enclosing the coils of the small intestine upon the right side of the abdomen, superiorly, and upon the left side of the abdominal cavity, the ascending colon and the descending colon being slightly overlapped by the coils of the small intestine.

The *cecum,* with the vermiform appendix, occupies the right iliac and the hypogastric regions. The cecum, comprising the blind extremity of the great intestine, and which lies inferiorly to the ileocecal valve, is approximately three inches in diameter and two and one-half inches in length. The *vermiform appendix* is a slender appendage of the cecum, usually approximately four inches in length and one-fourth inch in diameter, which is commonly given off from the postero-internal surface of the cecum.

surface of the liver which is situated external to the gall bladder. In a subsequent paragraph it will be shown that the anatomical relations of this portion of the large intestine to the right kidney and to the gall bladder may be utilized in the differential diagnosis of tumors springing from these structures.

The *transverse colon* crosses the abdominal cavity, descending from the right hypochondriac region into the upper portion of the umbilical region; thence ascending obliquely into the left hypochondriac region to the inferior extremity of the spleen to form the splenic flexure of the colon. The transverse colon, approximately twenty inches in length, is attached to the posterior abdominal wall by a broad fold of peritoneum, the transverse mesocolon, and it is the most freely mobile portion of the large intestine. Anteriorly the transverse colon is in apposition with the anterior abdominal wall, the great omentum alone intervening between the two structures. Posteriorly the transverse colon is in relation with the second or descending portion of the duodenum, and with coils of the small intestine. The transverse colon and transverse mesocolon serve in some sort as a dividing factor in the abdominal cavity, the liver, gall bladder, stomach, and spleen lying above this portion of the great intestine; and the coils of the small intestine occupying a position below its level.

The *descending colon*, commencing at the splenic flexure, passes vertically downward in the left lumbar and left iliac regions, to terminate in the sigmoid flexure in the hypogastric region. The descending colon is approximately nine inches in length; it is in relation anteriorly and laterally with the abdominal walls; and posteriorly it is in relation with the outer border of the left kidney and with the muscles forming the posterior abdominal wall.

The *sigmoid flexure* is approximately thirteen inches in length, commencing at the level of the iliac crest and terminating at the brim of the true pelvis in the rectum opposite the left sacro-iliac joint. The sigmoid flexure is the narrowest portion of the large intestine. When empty and collapsed, it falls into the rectovesical or rectovaginal pouch; but when distended with fecal accumulation or with gas it mounts up into the abdominal cavity, occasionally reaching the level of the umbilicus. The sigmoid flexure is attached to the posterior abdominal wall by a fold of peritoneum, the mesosigmoid, which permits this portion of the large intestine to enjoy a considerable degree of mobility. As a result of this free mobility the sigmoid flexure is occasionally the site of volvulus, an accident which is favored in many cases

by elongation of the mesosigmoid from the weight of accum
tion of fecal material in chronic constipation.

The *rectum*, the terminal portion of the large intestine, is
proximately nine inches in length. Commencing at the brim
the true pelvis at the left sacro-iliac joint, it passes obliqu
downward and inward to reach the third sacral joint, whence
continues its course directly downward in the median line of t
pelvis to a point one inch below the tip of the coccyx, where
turns abruptly backward to form the anal canal.

Physical Examination.—*Inspection.*—The undistended large i
testine, in the normal subject with abdominal walls in which th
musculature is not impaired, gives no clue to its presence upon in
spection of the abdomen. In the presence of pronounced gaseous distention of the colon, however, there is frequently a visible tumefaction in the right and left lumbar and in the hypogastric regions, which corresponds to the course of the ascending colon, the descending colon, and the sigmoid flexure, which in the state of gaseous distention mounts upward into the abdominal cavity proper. Gaseous distention of the transverse colon produces a protrusion in the umbilical region just above the umbilicus.

A circumscribed protrusion observed over the course of the colon, which persists and is demonstrable upon consecutive examinations, is apt to be due to a solid tumor of the colon. In such event it is attended by local tenderness upon palpation as a result of coincident peritoneal inflammation. In other instances a protrusion along the distribution of the colon is due to fecal impaction, in which event the tumefaction is transient and is relieved by thorough catharsis.

Palpation.—In the palpation of a tube of such extensive distribution and one which possesses such a variety of anatomical relations in different portions of its distribution, it is evident that a maneuver which is applicable to one portion of the tube is
not applicable in other portions. As a result of the changes in
lirection of the colon with reference to the different diameters of
he abdominal cavity, it is necessary to palpate different portions
f the tube in different planes.

In the practice of palpation of the *cecum* the examiner, seated
the right side of the patient, applies the right hand over the
d of the gut in the right lumbar and hypogastric regions and
adually sinks the fingers of the hand downward with a rolling
vement, endeavoring in this manner to outline the cecum. The
rgling murmur which is frequently set up by this maneuver

possesses no untoward significance. During this examination an attempt should be made to palpate the vermiform appendix, which is often to be felt along a line extending from McBurney's point toward the symphysis pubis as a cord about the size of a lead pencil and about the length of the little finger. A very serviceable method of palpating for the appendix consists in applying the finger-tips of both hands to the abdominal wall in the umbilical region, just to the right and a little below the umbilicus, and exerting pressure and at the same time moving the hands downward and outward over the abdominal wall toward the right anterior superior iliac spine. In this maneuver there is often felt under the fingers the small outline of the appendix.

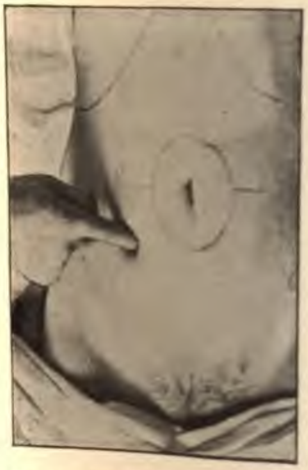


Fig. 235.—Indicating the point to seek for appendix tenderness. (From Crossen.)

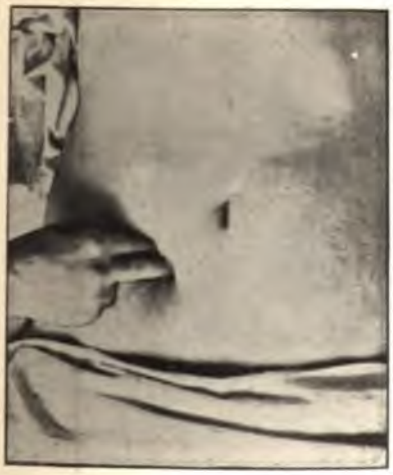


Fig. 236.—Palpating for tenderness or a mass in the appendix region. (From Crossen.)

If tenderness is encountered in the region of the cecum and appendix, and this is the most valuable finding upon palpation of this region, it is necessary to employ more careful palpation. In this instance, the tip of the index finger should be sunk slowly into the abdominal wall in the attempt to more accurately localize the area of local tenderness.

Tenderness elicited over the cecum points to appendicitis, typhlitis, perityphlitis, paratyphlitis, tumor, or tuberculous ulceration of the intestine. In the case of typhlitis, perityphlitis or paratyphlitis, in addition to tenderness there is a distinct feeling of resistance to the palpating hand, and the tenderness is rather diffuse as compared to that of acute appendicitis which is rather closely circumscribed to McBurney's point or to a spot slightly below this point. In interpreting the significance of local tenderness elicited in this region, it is worth while to remember that tuberculous ulceration of the intestinal tract is particularly liable to manifest itself in the ileocecal region. In the case of intestinal carcinoma, it is occasionally possible in the sensitive area to detect nobby bosses upon the intestinal wall.

In palpation of the *ascending colon* the patient should in the first instance assume the dorsal decubitus with the knees drawn up and supported; and, if examination in this posture is not satisfactory, the patient should be placed in the left lateral decubitus in order to cause the coils of the small intestine to fall away toward the left side of the abdominal cavity, leaving the fixed ascending colon free to manual palpation.

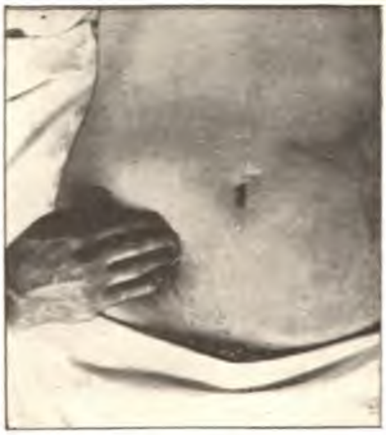


Fig. 237.—Palpating for the appendix itself, to determine whether or not there is any appreciable infiltration and thickening of it. When thickened, the appendix is felt as a small tender roll, deeply placed. (From Crossen.)

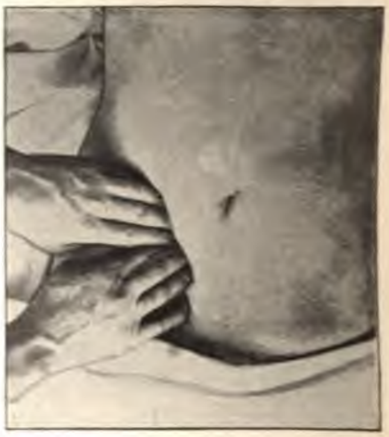


Fig. 238.—Another method of palpating the appendix. Beginning near the umbilicus, the fingers are carried in deeply and then brought slowly outward toward the anterior superior iliac spine. As the appendix passes under the examining fingers, it is felt as a small roll between the fingers and the posterior abdominal wall. (From Crossen.)

With the patient in the dorsal decubitus the examiner assumes a position by the right side of the patient and applies the finger-tips to the abdominal wall along the distribution of this portion of the intestine at right angles to the course of the ascending colon. By sinking the finger-tips into the abdominal wall and moving them outward with a sinking, rolling movement, the ascending colon frequently is felt between the finger-tips and the posterior abdominal wall, and the examiner may determine whether it is distended or contracted, whether its wall is free or is studded with nodulations, and whether it contains fecal masses. In this connection, it is well to remember that fecal accumulations are more frequently encountered in the cecum and ascending colon than elsewhere.

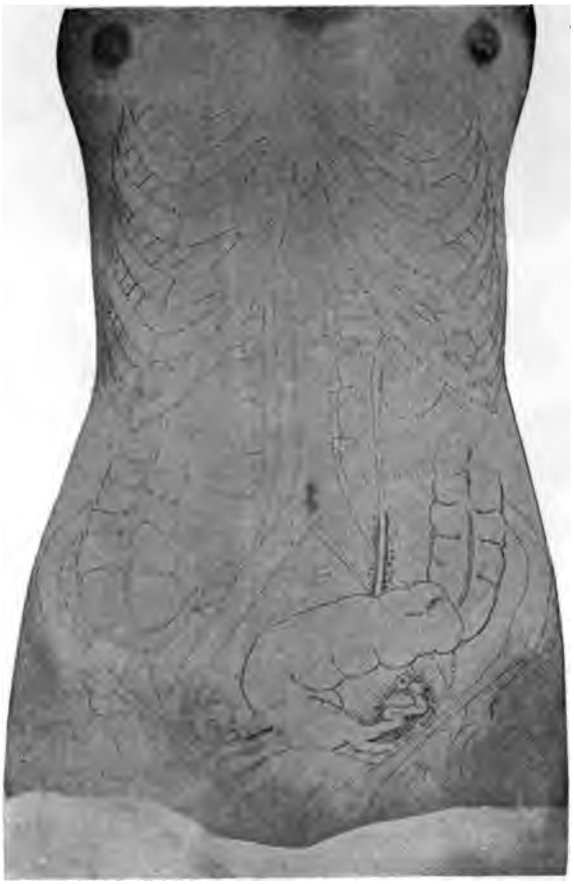


Fig. 239.—The left lower abdomen. The organs commonly affected, and the areas accordingly of particular interest, are indicated by the stippling. (From Crossen.)

If the palpation of the ascending colon in the dorsal decubitus is not entirely satisfactory, the maneuver should be repeated with the patient occupying the left lateral decubitus with the knees drawn up in order to relax the tension of the abdominal muscles. As the ascending colon is moored to the anterior face of the right kidney by areolar tissue, this portion of the intestinal tract is freely movable with the kidney; and in the presence of renal enlargement the ascending colon is pushed forward in front of the enlarged kidney.

During palpation of the *transverse colon* the patient assumes the dorsal decubitus while the examiner places both hands flat upon the abdomen in the umbilical region with the finger-tips extending somewhat above the level of the surface markings of the normal colon as it dips into and ascends from this region of the abdomen. The patient is directed to inspire deeply; and at the commencement of the following expiration the examiner makes downward pressure with the finger-tips and endeavors to determine the position and state of this portion of the intestine. In palpation of this section of the colon, as in the case of other portions, the hand should be applied to the abdominal surface at right angles to the course of the colon. Hence, if the transverse colon pursues an arched course with the convexity directed downward, as is very frequently the case, the right and left halves of this portion of the intestine must be palpated in different directions. While the normal transverse colon is encountered at the level of or above the umbilicus, in enteroptosis it may be encountered as low as the symphysis pubis.

The *descending colon* is palpated with the patient in the dorsal decubitus or in the right lateral decubitus, the technic of the examination corresponding in all its essentials to the method employed in palpation of the ascending colon.

Palpation of the *sigmoid flexure* is performed with the patient in the dorsal decubitus, the examiner, seated at the left side of the subject, placing the fingers of the right hand upon the abdomen at right angles to the course of the gut, rolling the intestine between the finger-tips and the iliac fossa, searching for tenderness, muscular rigidity, tumor, or fecal impaction. As a general rule impacted feces will readily pit upon pressure, whereas a solid tumor will fail to do so. Moderate tenderness over the sigmoid is not infrequently encountered in the course of diarrhea from any cause; and in dysentery of specific origin it is as much more pronounced.

Percussion.—As in the case of the small intestine, the findings upon percussion of the colon are variable and extremely difficul of correct and accurate interpretation. The note elicited u percussion of the colon containing gas is frankly tympaniti metallic, its quality and pitch varying with the tension of colonic walls. Yet it is practically impossible at the bedsi differentiate the percussion sounds elicited upon percussion of the stomach, the small intestine, and the colon with any degree of accuracy, as the quality and pitch of the sounds vary with variations in the tension of the walls of the various portions of the intestinal tract; and it is possible for coils of the small intestine, in spite of their lesser size and capacity to yield a tympanitic note quite analogous to that which is generated in the moderately distended colon when the coils of the small intestine are strongly distended with gas and their walls rendered very tense. The tympany of the transverse colon is differentiated from that of the stomach with most ease when the latter organ is partially filled with fluid. The presence of fecal accumulations in the various portions of the large intestine masks the tympanitic quality of the percussion note, yielding varying degrees of dullness upon percussion.

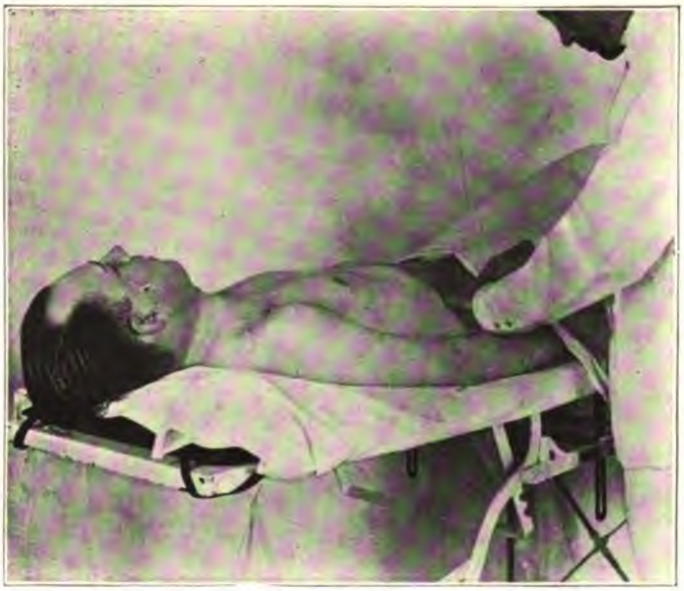


Fig. 240.—Palpation of ascending colon.

Auscultation.—The chief value of auscultation in connection with the examination of the large intestine is in auscultatory percussion in differentiating the gastric and the colonic tympany. Auscultation frequently reveals the presence of gurgling sounds over the cecum in the presence of typhoid fever, which are in no wise pathognomonic of this disease. Similarly, in the presence of severe diarrhea or dysentery gurgling is encountered over the sigmoid flexure.

Very rarely is the examiner able to detect upon auscultation a peritoneal friction sound in the presence of inflammation of the walls of the colon or in the presence of miliary tuberculosis or malignant disease of this tract.

The physical examination of the gastrointestinal tract should in all cases be abetted by the chemical and microscopical examination of the gastric contents and the feces, by illumination of the rectum; and when pathologic lesions of the tract are suspected, fluoroscopy should be practiced by a skilled roentgenologist.

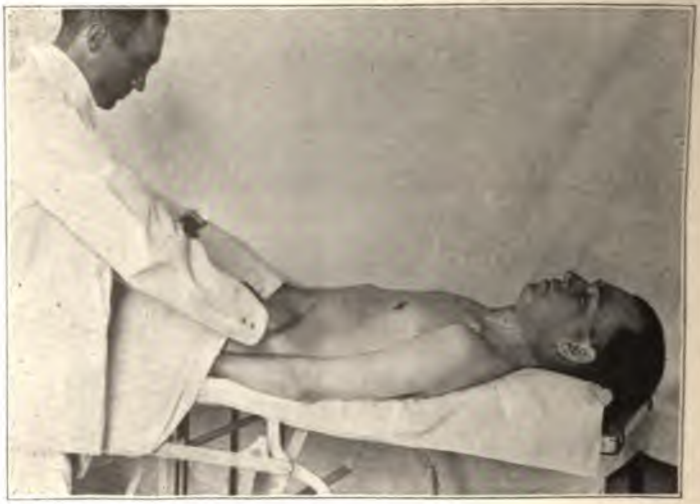


Fig. 241.—Palpation of the descending colon.

EXAMINATION OF THE PANCREAS

Clinical Anatomy.—The pancreas is an elongated, solid organ, comprising a head, neck, body, and tail, situated deeply in the epigastric region, its tail, however, extending into the left hypochrondriac region.

The pancreas in the adult subject is approximately six inches in length, and its location in the abdominal cavity corresponds to the level of the first and second lumbar vertebræ. The head of the pancreas is encircled by the second and third portions of the duodenum, the common bile duct intervening between the two structures, while the tail extends toward the left and is in relation with the hilum of the spleen.

The body of the pancreas is roughly prismatic in shape and possesses three surfaces, which respectively are directed anteriorly, posteriorly, and inferiorly. The anterior surface, covered by the peritoneum of the lesser sac, is in relation with the posterior wall of the stomach, the transverse colon, and a few coils of

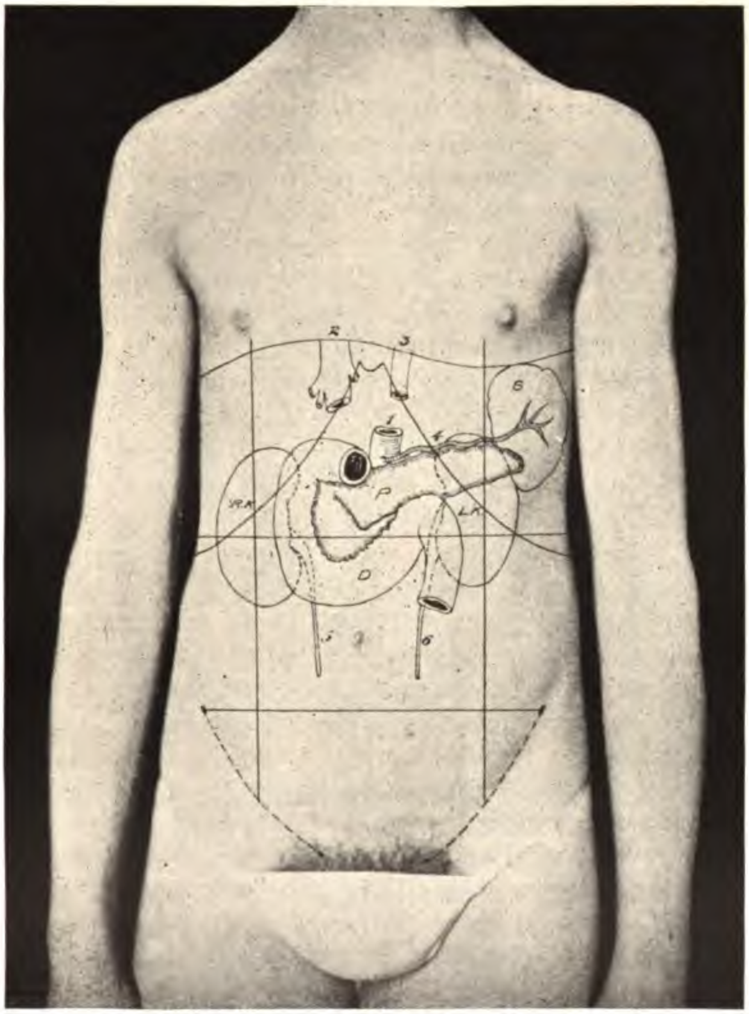


Fig. 242.—Relations of pancreas to adjacent viscera.

1, aorta; *2*, inferior vena cava; *3*, esophagus; *4*, splenic artery; *5* and *6*, ureters; *RK*, right kidney; *LK*, left kidney; *D*, duodenum; *P*, pancreas; *S*, spleen. (From Eisendrath.)

the small intestine. The posterior surface, devoid of peritoneal covering, rests upon the abdominal aorta, the inferior vena cava, the right crus of the diaphragm, the splenic vein, the left kidney, renal vessels, and suprarenal capsule, and the commencement of the superior mesenteric artery. The posterior aspect of the neck of the pancreas is in relation with the commencement of the portal vein. The inferior surface is in relation with the duodenojejunal flexure of the small intestine, a few coils of the jejunum, and its left extremity rests upon the splenic flexure of the colon. As a result of the intimate relation of the pancreas to so many important blood vessels, enlargements of the gland are not infrequently attended by vascular bruits due to narrowing of the lumen of one or more of these vascular trunks. Similarly, in malignant disease of the head of the pancreas, the common bile duct, intervening between this portion of the gland and the second portion of the duodenum is frequently compressed with the production of icterus, which may simulate that of hepatic disease.

The pancreatic secretion is conveyed to the second portion of the duodenum by the pancreatic duct of Wirsung, which empties its contents into this portion of the intestine by a common orifice with the common bile duct. Occasionally the pancreas possesses an accessory excretory duct, the duct of Santorini, which empties its contents into the second portion of the duodenum approximately one inch above the level of the orifice of the principal pancreatic duct. Stenosis of the pancreatic duct usually leads to cystic enlargement of the pancreas; and the entrance of infective bacteria from the intestine by way of the pancreatic duct may induce suppurative disease of the pancreas. Malignant disease of the pancreas usually involves the head of the gland to the exclusion of other portions, and carcinoma comes first in frequency among pancreatic morbid growths, although sarcoma and adenoma also attack the gland.

The pancreas crosses the lower portion of the epigastric region, the tail of the gland extending approximately two inches into the left hypochondriac region, and the lower extremity of the head of the gland dipping into the upper portion of the umbilical region. The lowest point of the gland occupies a position approximately three inches above the umbilicus.

Physical Examination.—Owing to the profound situation of pancreas in the abdominal cavity and as a result of its ra flatly prismatic conformation, physical examination of the o practically resolves itself into the practice of palpation, occasionally aided and abetted by auscultation.

The examiner will very rarely be able to palpate the normal pancreas, save possibly in the emaciated subject with diastasis of the rectus muscles. Leube and Ewald have been able to palpate the normal organ when the stomach and intestine were empty; but as a rule, the examiner may anticipate considerable difficulty in palpation of the normal pancreas.

When a mass is encountered in the deeper portion of the lower epigastric or upper umbilical region, it may be due to a solid tumor or a cyst of the pancreas; and pancreatic solid tumors in the vast majority of cases are carcinomatous.

A tumor arising from the pancreas is apt to be mistaken for a tumor of the pylorus, a distended gall bladder, aortic aneurysm, or a tumor of the transverse colon. A pyloric tumor is always more superficial than is a pancreatic growth. Moreover, a tumor at the pylorus is freely movable, in marked contrast to the immobility of pancreatic tumors, and pyloric tumors are prone to produce pyloric stenosis with consequent gastrectasis. A distended gall bladder exhibits lateral mobility; it is more superficial than is a tumor of the pancreas; and it is not accompanied by glycosuria and fatty stools as are pancreatic growths very frequently.

A pancreatic tumor may be elevated with each pulsation of the abdominal aorta, simulating aortic aneurysm; but while the pulsation of aortic aneurysm is expansile and is exercised in all directions, that of a superjacent pancreatic tumor is not expansile, the mass being simply elevated with each pulsation of the subjacent aorta.

A tumor of the transverse colon is very superficial; it is freely mobile; it is prone to cause constipation; and blood is frequently demonstrable in the stools with such growths.

A pancreatic cyst occasionally acquires a considerable size, and manifests itself upon palpation as a resistant, fluctuating tumor. There is danger of confounding them with hydatid cysts of the liver, or with ovarian cysts when these attain huge dimensions. In the differential diagnosis of pancreatic and ovarian cysts, Kuester has directed attention to the fact that in the case of pancreatic cyst the inferior border of the cyst is always separated from the symphysis pubis by a considerable zone of intestinal tympany, whereas in the case of a cyst springing from the ovary this zone of tympany in the hypogastric region is absent. In the differentiation of pancreatic cysts and hydatid cysts of hepatic

CHAPTER XXV

EXAMINATION OF THE LIVER AND GALL BLADDER

Clinical Anatomy.—The liver, the largest gland of the body, occupies the upper right quadrant of the abdominal cavity, lying in the right hypochondriac region, the larger portion of the epigastric region, the thin extremity of the left lobe extending into the left hypochondriac region, and the inferior extremity of the right lobe invading the right lumbar region for a short distance. The liver is roughly wedge-shaped, with the wide base directed toward the right, and the thin sharp edge of the wedge directed toward the left side of the abdomen. The normal adult liver measures eight to nine inches transversely, six to seven inches vertically at the base of the wedge, and four to five inches anteroposteriorly at a point on a level with the upper border of the right kidney.

The liver is divided into two unequal portions, the right and left lobes, by the falciform ligament and longitudinal fissure, the right lobe greatly exceeding the left lobe in size. At the point where the falciform ligament joins the inferior margin of the liver there is a small notch, the umbilical notch, which is situated at the level of the ninth right costal cartilage one inch to the right of the median line. Slightly beyond this notch the liver presents a second notch, in which is lodged the fundus of the gall bladder, corresponding to the junction of the ninth rib and the right border of the rectus muscle.

The *superior surface* of the liver, smooth and convex, is closely applied to the concave right vault of the diaphragm. Upon its central portion the superior surface of the liver presents a shallow depression, the *cardiac depression*, corresponding to the position of the heart upon the superior surface of the diaphragm.

The *anterior surface* of the liver is applied to the inferior surface of the diaphragm, which separates it from the lower ribs and their cartilages upon the right and left sides, while in the median line it comes into direct contact with the anterior abdominal wall in the subcostal angle.

The *right* and *posterior* surfaces of the liver are in contact with the inferior aspect of the diaphragm, which separates the liver from the right pleural cavity and the lower border of lung.

The *inferior surface* of the liver, directed somewhat p and toward the left, is in relation with the stomach, th flexure of the colon, the right kidney and suprarenal the second portion of the duodenum and the gall bladder

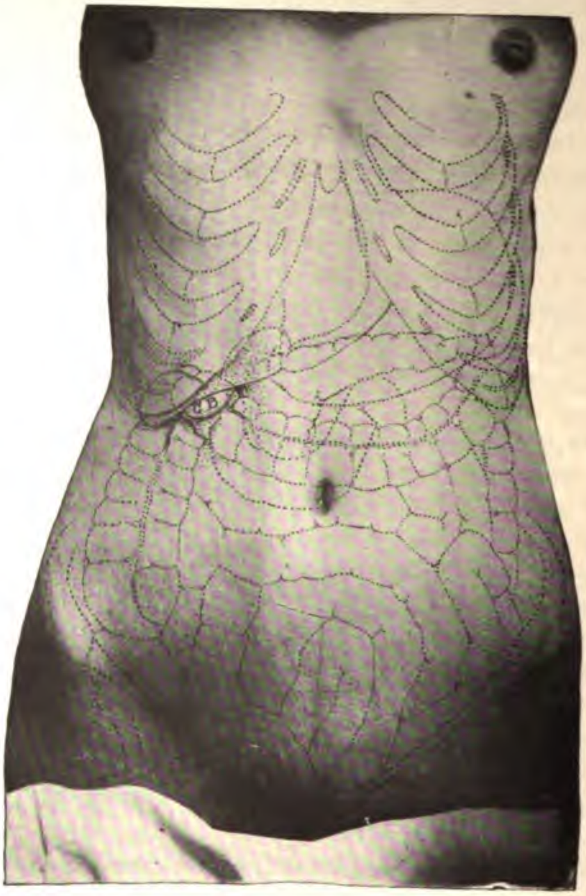


Fig. 243.—The right upper abdomen. The site of the gall bladder, the area of pa interest in this region is indicated by the letters *G.B.* (From Crossen.)

The hepatic parenchyma is enclosed in a fibrous capsule, w is in turn invested with peritoneum with the exception of a li portion of the posterior surface of the liver, constituting "bare area" of the organ, which is united to the inferior su of the diaphragm by areolar tissue. During the course of in matory disease of the organ the serous investment occasionally becomes rugose and roughened with the production of a peritoneal friction sound upon the respiratory movements of the organ or upon manipulation of the abdominal wall in the hepatic region.

In addition to the coronary ligament, the liver is retained in position by the falciform ligament, the round ligament, and two lateral ligaments. The falciform ligament is a broad fold of peritoneum which is attached on the one hand to the inferior surface of the liver at the umbilical notch, and upon the other hand to the inferior surface of the diaphragm and to the posterior surface of the sheath of the rectus muscle as low as the umbilicus. The round ligament is a small fibrous cord representing the remnants of the umbilical vein after it has undergone occlusion. It passes from the umbilicus in the free margin of the falciform ligament to the umbilical notch of the liver, whence it passes along the umbilical fissure upon the inferior surface of the liver, to be continued upon the posterior surface of the organ as the impervious ductus venosus up to the inferior vena cava. The lateral ligaments attach the extremities of the liver to the inferior surface of the diaphragm, being largely composed of the lateral reflections of the coronary ligament.

The liver is supported in the abdominal cavity partially by virtue of its ligamentous connections with the abdominal walls, partially by an areolar attachment to the inferior aspect of the diaphragm in the "bare area" of the posterior surface of the organ, and partially by the pressure which is exerted upon the organ by the other abdominal viscera. In the event of relaxation of the hepatic ligaments, the liver falls downward to occupy a lower level in the abdominal cavity; and in the presence of general visceroptosis the liver shares in the descent of the abdominal viscera.

The liver is movable within certain limits. Owing to its accurate apposition with the inferior surface of the diaphragm, it participates in the respiratory excursions of this muscle, descending during inspiration and ascending during expiration. The weight of a right-sided pleural effusion or the pressure exerted in right pneumothorax is equally capable of depressing the liver and causing its sharp inferior border to project below the right costal arch.

The habitual wearing of tight clothing exercises a distinct influence upon the form and the position of the liver. There is under these circumstances a progressive flattening of the superior surface of the organ with a concomitant lengthening of the right lateral surface upon which is frequently noted a constriction corresponding to the edge of the costal arch, the tapering lower extremity of the right lobe in this state constituting Riedel's lobe.

The portal vein and hepatic artery enter the transverse fissure of the liver between the layers of the gastrohepatic omentum, which unites the liver with the stomach. The hepatic duct, in close relation with these vessels, passes downward from the transverse fissure between the layers of the same omentum.

The *gall bladder*, the reservoir for the bile, reposes in a shallow depression upon the inferior surface of the right lobe of the liver, the tip of the fundus meeting the anterior abdominal wall in the notch upon the inferior border of the liver situated at the junction of the ninth right costal cartilage with the outer border of the right rectus muscle.

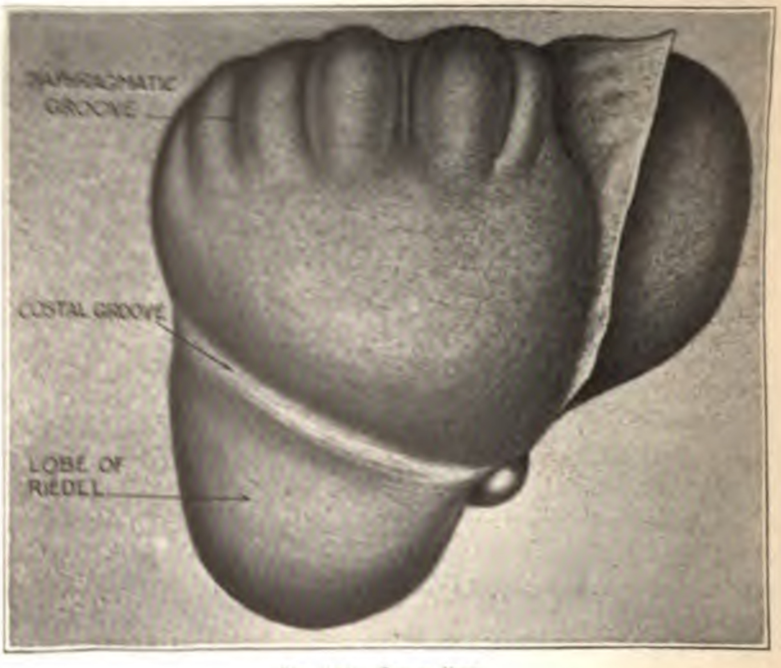


Fig. 244.—Corset liver.

The gall bladder is a pear-shaped sac which is approximately four inches in length and one and one-half inches in diameter when moderately distended. From the fundus of the sac, the body tapers to form the neck, which in turn terminates in the cystic duct, which unites with the hepatic duct to form the common bile duct.

The common bile duct, the common excretory duct of the liver and the gall bladder is approximately three inches in length. It descends in the folds of the lesser omentum, passing behind the first portion of the duodenum, and passing between the head of the pancreas and the descending portion of the duodenum, it empties its contents into the second portion of the duodenum usually by a common orifice with the pancreatic duct. As a result of the relations of the common bile duct to the pancreatic head and the descending portion of the duodenum, stenosis of the duct frequently ensues in the event of malignant disease of the head of the pancreas with the production of hepatic enlargement and icterus. Stenosis of the duct may also ensue as the result of the lodgment of gallstones along its course. In the event of the impaction of a large gallstone at the common orifice of the common bile duct and of the pancreatic duct, it has been demonstrated that the pressure of the bile in the common duct is capable of overcoming the pressure in the pancreatic duct, with the result that bile accumulates in the pancreatic duct, perhaps with the production of inflammation of the pancreas.

Surface Topography.—The *superior border* of the liver corresponds to the level of the lower border of the sixth rib in the midclavicular line, the lower border of the eighth rib in the midaxillary line, and the lower border of the tenth rib in the scapular line.

The *inferior border* of the liver corresponds to a line drawn downward and toward the right from the lower border of the sixth rib in the left midclavicular line, the point upon the surface corresponding to the left extremity of the organ, the line crossing the left costal arch at the eighth costal cartilage, the median line four inches below the ensiform cartilage, the right costal arch at the ninth costal cartilage, the lower border of the tenth rib in the midaxillary line, and the lower border of the eleventh rib in the scapular line.

Physical Examination.—*Inspection.*—Inspection of the hepatic region of the abdomen is practiced to the best advantage with the subject in the upright or sitting posture; and the examination should be conducted under oblique illumination of the abdominal wall. The examiner assumes a position to the right side and a little distance in advance of the subject, and studies the contour of the right hypochondriac region during deep inspiration on the

part of the patient. In the normal adult subject this regi
the abdomen will not be observed to present any departure
the aspect of the opposite side so long as the liver is of n
volume and is maintained in its normal habitat. In the chil
the contrary, in whom the liver is uncommonly large in pr
tion to the dimensions of the adult organ, it is not infrequ
observed that a furrow rises and falls with the movemen
expiration and inspiration, which indeed may descend to the
of the umbilicus in this class of subjects. When this furr
observed in the adult male, it is significant of downward dis
ment of the liver or of enlargement of the organ.

Any extensive degree of enlargement of the liver is manif
by undue prominence of the right costal margin with fullne
the right hypochondriac and the epigastric regions, whic
cases of excessive hepatic enlargement may extend to the e
anterior abdominal wall. Under these circumstances it is,
rule, easy to perceive an inspiratory depression and an expir
elevation of the inferior border of the organ. It is very impo
to observe that in the case of flaring of the lower right c
arch as a result of hepatic enlargements the intercostal space
not obliterated, and that they can be readily palpated throug
their entire extent; whereas in the event of flaring of the c
arch as the result of an extensive pleural effusion the inters
are obliterated. In the case of pleurisy with effusion the i
thoracic excess of pressure is exerted in a direction down
and outward, whence it follows that the lower costal arch is
duly prominent, while the ribs and intercostal spaces confor
their normal courses; whereas in the case of protrusion of
costal arch from hepatic enlargement the intraabdominal pre
is exerted in a direction upward and forward with the result
the lower ribs undergo an abnormal torsion, their internal
faces becoming inferior and their external surfaces being dir
superiorly.

In interpreting respiratory mobility of tumors arising in
upper abdominal cavity, the examiner should bear in mind
growths of the liver and of the spleen alone possess a true
piratory mobility; that respiratory excursions are always
pronounced upon the side of the liver on account of its mor
timate relation to a more extensive surface of the inferior a
of the diaphragm; and that respiratory movement is only
parted to growths of the stomach or intestine when these
have contracted adhesions with the liver or with the spleen,

result in the transmission of false respiratory movements to these growths.

The detection of mobile excursions of the inferior border of the liver below the right costal arch does not, however, point invariably to enlargement of the organ. The abnormally low position of this sharp margin of the organ may be due to displacement downward of the liver by an increase in the intrathoracic pressure which is exerted upon the superior aspect of the diaphragm, or it may be due to a falling of the liver as the result of relaxation of its suspensory ligaments.

Of these two factors, the former operates with the greater frequency in the production of depression of the liver. In the presence of extensive effusion into the right pleural sac in serofibrinous pleurisy, in the event of the development of hydro- or pyopneumothorax, in the course of an extensive pericardial effusion, or as the result of the progressive development of neoplasms in the mediastinum, the liver is forced downward in the abdominal cavity and its inferior border becomes perceptible below the right costal arch. Relaxation of the hepatic ligaments, permitting the liver to fall to an abnormally low level in the abdominal cavity, is encountered most frequently in multiparæ in whom successive pregnancies have caused general relaxation of the abdominal walls, and hepatoptosis in these cases is often merely part and parcel of general visceroptosis.

Only occasionally is it possible upon inspection of the respiratory excursions of an enlarged liver to detect local protrusions of the inferior border of the organ, which are in some instances produced by a solid tumor of the organ, in other cases are due to abscess, and yet again to cystic tumors of the organ. Also, occasionally in the course of hepatic abscess or neoplasm adhesions form between the liver and the abdominal wall, with the consequent participation of the latter in the morbid state of the liver. In these instances one occasionally encounters in the hepatic area a circumscribed protrusion of the ventral abdominal wall which may be the site of fluctuation or which may be discolored and sensitive to pressure.

A local protrusion below the right costal arch at the junction of the ninth right costal cartilage with the outer border of the right rectus muscle is frequently encountered in distention of the gall bladder with bile, serous fluid, or purulent material. Similarly, in the presence of carcinoma of the gall bladder the nodular rigid wall of the viscus frequently causes circumscribed prominence in this area. A distended gall bladder retains in general the original contour of this viscus, and presents upon inspection a circumscribed pyriform elevation of the abdominal wall. The fundus of the gall bladder under these circumstances may be encountered as low as the umbilicus.

When a large protrusion is encountered in the lower anterior and lateral portions of the hepatic area, the examiner should bear in mind the possibility of the existence of a corset liver in the female subject. This deformity of the liver may extend even to the level of the iliac crest; and when it is overlapped by coils of intestine, it may readily be mistaken for a new growth springing from the right kidney or the ascending colon.

A visible systolic *pulsation* of the liver is occasionally noted in connection with tricuspid regurgitation, though usually the pulsation requires bimanual palpation for its recognition. Similarly, in the case of the enlarged liver, the systolic pulsation of the subjacent abdominal aorta may be imparted to the liver, when it is manifested in the form of a simple rising and falling of the organ and not in the form of a true expansile pulsation. A transmitted impulse conveyed to the liver by the impact of an over-acting heart should not be mistaken for a true pulsation of the liver.

Palpation.—In practicing palpation of the inferior hepatic border the patient should assume the dorsal decubitus with the knees drawn up and supported. The examiner, seated upon the right side of the patient, should place the finger-tips of the right hand upon the abdominal surface immediately below the right costal arch, and by gradually sinking the finger-tips into the abdominal wall, should seek for the lower margin of the liver. If it be found that the liver projects below the costal margin, the examiner should apply both hands flat upon the abdomen, and by downward pressure cause the finger-tips to glide over the exposed portion of the liver, searching for tenderness, any abnormality in contour, and investigating the consistence of the organ.

In the normal adult subject the inferior border of the liver will not be encountered below the costal arch in the male subject. In the female subject, on the contrary, as the result of the habitual use of clothing which constricts the lower portion of the thorax, the inferior border of the liver not infrequently occupies a position below the right costal margin. In multiparæ the relaxation of the abdominal walls and of the suspensory ligaments of the liver tends further to produce lowering of the liver in these subjects. In young children the inferior border of the liver is very frequently encountered below the costal margin.

If the inferior margin of the liver is not encountered below the costal margin, the examiner should place the finger-tips of both hands upon the abdominal surface just below the costal arch and should press inward and upward beneath the costal arch as far as is possible. The subject is directed to inspire deeply, when during inspiration the liver will be felt to descend with the descent of the diaphragm and to become palpable at the completion of inspiration. By the procedure first described an enlarged liver or a depressed liver will be revealed projecting below the costal arch; while, by the second maneuver, a normal or contracted liver may frequently be palpated.

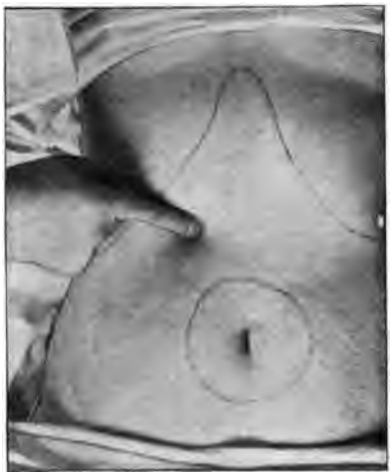


Fig. 245.—Indicating the site for tenderness or a mass due to disease of the gall bladder. It may be found anywhere from the point indicated downward and outward to the margin of the ribs on the right side. (From Crossen.)

The facility with which the liver is palpated is markedly influenced by the state of the abdominal walls. An excessive deposition of fat in the walls impedes and occasionally renders quite unsatisfactory the exploration of the liver by palpation. In the multipara, on the contrary, with relaxation of the abdominal walls and with possible diastasis of the recti, the manual exploration of the organ is rendered so much the more easy.

Similarly, the presence of gas or fluid in the intestinal tract or in the abdominal cavity is a frequent cause of confusion in palpation of the liver. In the presence of ascites, which is so often due to hepatic affections and renders a careful palpation of the organ so desirable, the examiner is occasionally able to reach the inferior border of the liver by exercising a few rather sharp, jerky palpating movements toward the lower border of the organ, which by crowding the fluid aside enables him to reach the liver. In this class of patients the examination is further facilitated by placing the patient in the left lateral decubitus or in the genupectoral position. In the case of ascites of suspected hepatic origin, the fluid should be evacuated from the abdominal cavity, whereupon palpation of the liver becomes very easy and is apt to reveal some very striking findings.

Enlargement of the liver accompanies the acute infectious fevers, fatty infiltration of the organ, chronic passive congestion of the organ, and amyloid disease of the liver. Similarly, hepatic enlargement attends Weil's disease, hepatic abscess, carcinoma or gumma of the organ, leukemia, hypertrophic hepatic cirrhosis, echinococcus cyst, and Banti's disease.

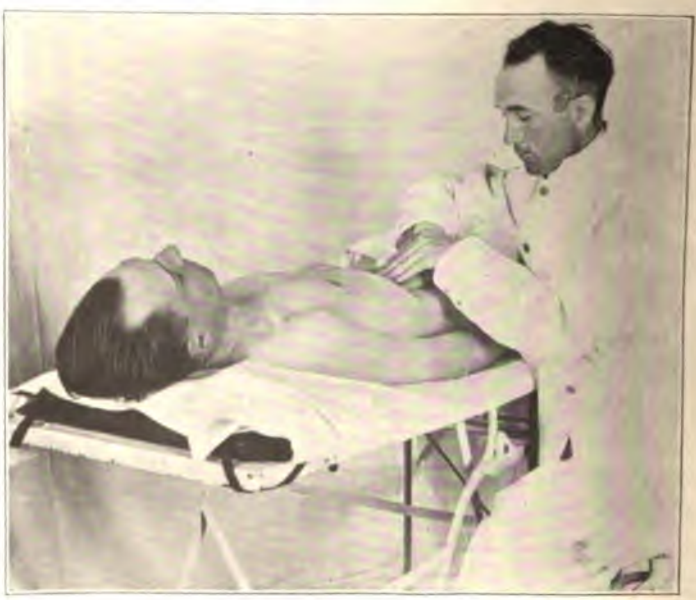


Fig. 246.—Palpation of liver.

In every case in which enlargement of the liver is suggested by the finding of a palpable inferior margin of the liver, the examiner should continue the examination in the effort to determine the state of the surface of the liver, the consistence of the organ, the presence of sensibility of the liver, and the respiratory mobility of the mass.

When the inferior border of the liver is appreciated by the palpating hand, it may be found to be smooth or to be irregular and the site of nodular elevations and depressions. When a smooth surface is encountered, the examiner should proceed to identify it with the liver by establishing upon its surface the two notches of the inferior hepatic border, the one approximately one inch to the right of the median line and the second at the outer border of the right rectus muscle.

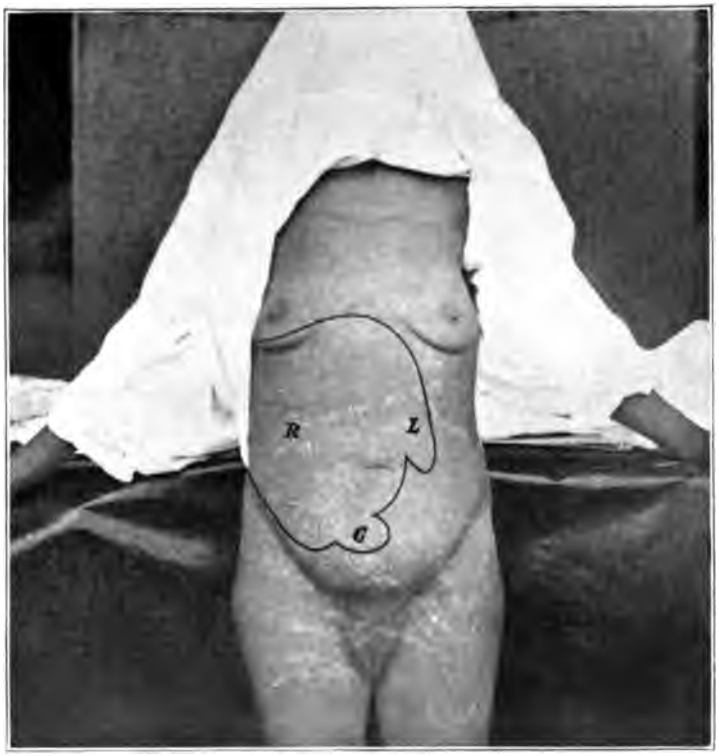


Fig. 247.—Hepatic enlargement due to carcinoma of head of pancreas. *R*, right lobe of liver; *L*, left lobe of liver; *G*, distended gall bladder. (From Eisendrath.)

An irregular, nodular hepatic surface is encountered in carcinomatous infiltration of the liver, in atrophy of the organ of syphilitic or nonspecific origin, and in echinococcus disease of the organ. The nodules of carcinoma present a central umbilication, which can occasionally be appreciated in palpation of the liver through a thin abdominal wall. The nodules of the syphilitic liver are uniformly small, whereas the irregularity of the liver in the case of echinococcus disease takes the form of a few protrusions of relatively extensive dimensions.

The palpable *consistence* of the liver occasionally gives a clue to the nature of the cause of the enlargement. The fatty liver possesses a consistance scarcely altered from that of the normal organ, while that of chronic passive congestion scarcely offers any more resistance. In the case of amyloid disease of the liver, on the contrary, the organ frequently attains a woody hardness. Carcinomatous infiltration of the liver produces a liver of hard consistence. In the case of hepatic abscess and echinococcus cyst the consistence of the mass is softened and fluctuation is frequently obtained under

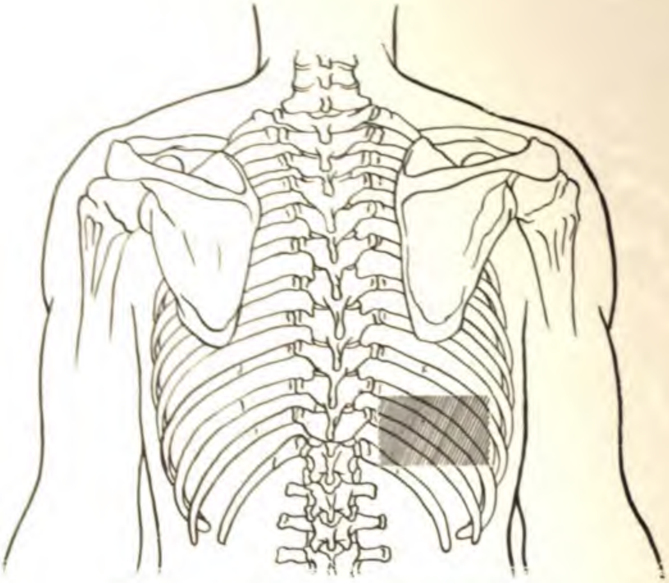


Fig. 248.—Dorsal pressure point in cholelithiasis.

these circumstances. Frerichs has found echinococcus disease of the liver, however, associated with the development of multilocular cysts of the organ, in which the liver possessed a consistence almost cartilaginous, suggesting strongly carcinomatous infiltration of the organ. Jaccoud has encountered fluctuation of the liver in certain cases of hypertrophic cirrhosis of the organ.

Sensibility of the liver, manifested by pain upon pressure, may be diffuse or may be circumscribed to certain definite regions of the abdomen. Diffuse sensibility of the liver is encountered in fatty degeneration and chronic passive congestion of the organ, in hypertrophic cirrhosis, in hepatic abscess and carcinoma, and in inflammatory disease involving the peritoneal covering of the organ in perihepatitis. Circumscribed sensibility is usually significant of affections of the gall bladder, the tenderness of an acutely inflamed gall bladder manifesting itself upon pressure over the fundus of this viscus at the junction of the ninth right costal cartilage and the outer border of the right rectus muscle. Similarly cholelithiasis is attended besides by a tender point upon pressure immediately to the right side of the twelfth dorsal vertebra posteriorly.

The respiratory *mobility* of the inferior border of the liver, which was described under inspection, can be readily detected upon palpation of the inferior border of the organ. During inspiration this border of the organ descends, only to ascend to a corresponding degree during expiration. The respiratory mobility of masses of hepatic origin is a valuable sign in differentiation from similar masses springing from the stomach, intestine, kidney, or pancreas.

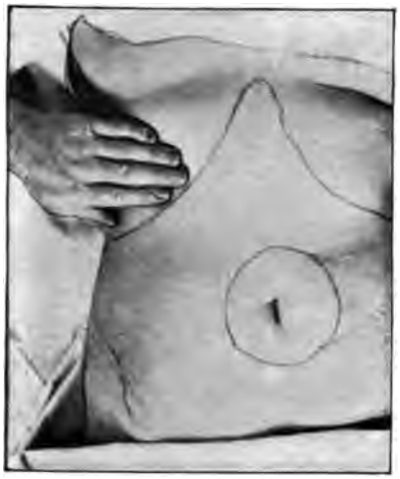


Fig. 249.—Palpating for general tenderness of the liver. (From Crossen.)

But while the growths springing from the liver and from the spleen alone among intraabdominal growths possess a true respiratory mobility, it does not follow that all hepatic growths are mobile. The presence of extreme tympanites or ascites impairs or prevents the respiratory mobility of certain hepatic enlargements. Similarly, when an enlarged liver has established firm adhesions with the abdominal walls, respiratory mobility of the organ is abolished. Also as inflammatory disease of the liver is very frequently ac-inferior border of the liver, in such a fashion that the distended gall bladder assumes the form of a mass independent of the liver upon palpation and percussion. In addition to respiratory mobility, the distended gall bladder presents a distinct lateral mobility upon palpation.

Occasionally when the gall bladder contains multiple stones, a sensation of crepitation is appreciated upon palpation of the fundus of the viscus.

The palpation of the hepatic region should not be considered complete until the examiner has practiced bimanual palpation of the liver with the view of detecting true expansile pulsation of the organ in the presence of tricuspid insufficiency.

Percussion.—In practicing percussion of the hepatic area the student succeeds in establishing two gradations of dullness due to the presence of the liver in the upper and right segment of the abdomen below the lower ribs and intercostal spaces. These areas constitute respectively the areas of hepatic dullness and of hepatic flatness.

The Areas of Hepatic Dullness and Flatness.—Upon percussing downward over the surface of the thorax and abdomen in the midclavicular, midaxillary, and scapular lines from an area of frank vesicular resonance, the percussion note will become impaired or dull when the point is reached where the superior limit of the liver is covered by the pulmonary tissues. The point of change in the quality of the note indicates the upper limits of the *area of hepatic dullness.* The superior limits of this area are encountered at the upper border of the fifth rib in the right midclavicular line, the seventh intercostal space in the right midaxillary line, and at the eighth intercostal space in the right scapular line.

Upon continuing the percussion downward along these lines, substituting light for forcible percussion, a point is reached in which the dullness gives place to flatness, indicating the upper limit of the region where the liver is in direct contact with the abdominal wall, the superior limit of the *area of hepatic flatness.* The superior limits of this area are found in the normal adult subject at the lower border of the sixth rib in the right midclavicular line of the eighth rib in the right midaxillary line, and of the tenth rib in the right scapular line.

If now the percussion is continued downward along the same lines, the flat note will be replaced by intestinal tympany when the inferior limit of the liver is attained. The points of change indicating the inferior limit of the area of hepatic flatness are encountered at the ninth rib in the right midclavicular line, the tenth rib in the right midaxillary line, while in the right scapular line the flatness of the liver is continuous with that produced by the kidney.

The inferior limit of the area of hepatic flatness in the epigastric region lies three to four inches below the ensiform cartilage. Thus, it is observed that the areas of hepatic dullness and flatness extend downward and toward the right; that posteriorly they are continuous with the flatness of the right kidney; and that anteriorly they blend with the right border of the area of cardiac dullness.

In pathologic states the hepatic dullness may be abolished, may be diminished in extent, may be increased in extent, or the area of dullness as a whole may be displaced from its normal site.

Absence of hepatic dullness upon percussion of the hepatic area is significant of falling of the liver, in which event the dullness of this organ is replaced by intestinal tympany. This state, which is usually encountered in multiparæ as a result of extreme relaxation of the suspensory ligaments of the liver, may permit the liver to occupy the iliac fossa, constituting the "floating liver" of Cantani. In this abnormal situation the liver could readily be confused with an abdominal tumor. In this connection it is to be remembered that the displaced liver occupies in the main the right half of the abdominal cavity; that its superior surface is convex, smooth, and rounded; and that palpation of its inferior sharp margin reveals the presence of the two notches which exist upon this border of the organ. Winckler and Sutugin have succeeded, during palpation of a displaced liver, in outlining the suspensory ligaments of the organ and in tracing them to their connections with the lateral abdominal walls. The displaced liver can usually be restored to its normal habitat by manual manipulation, whereupon the intestinal tympany is replaced by hepatic dullness in that region.

When tympanites is present in a pronounced degree, the liver is not infrequently crowded upward into the right vault of the diaphragm and percussion of the hepatic area under these circumstances yields only mediocre dullness in comparison with the excessive tympanicity of the percussion note over adjacent portions of the abdomen. Similarly when the liver and inferior diaphragmatic surface have contracted adhesions which exert traction upon the superior surface of the organ, the area of hepatic dullness is very much restricted and occasionally is outlined with difficulty if indeed at all. Also in transposition of the viscera, when the spleen occupies the right upper quadrant of the abdominal cavity and the liver lies in the upper left quadrant, the area of hepatic dullness is defective. As a rule in these cases the thoracic viscera are also transposed, the cardiac apex lying upon the right side of the sternum; but Mosler has encountered cases in which the transposition of the viscera was limited solely to the liver and the spleen.

Diminution of hepatic dullness is encountered in all cases of diminution in the dimensions of the liver; but it would be erroneous to immediately conclude that the liver has diminished in volume in every case in which there is a restriction of the area of hepatic dullness. Before definite conclusions may be formulated in respect to the dimensions of the liver, it is necessary to establish the absence of certain conditions which may simulate a diminution in the volume of the liver. Occasionally the large intestine insinuates itself in front of the lower border of the liver, simulating a diminution in volume of this organ. In these cases it is necessary to endeavor by forcible compression with the pleximeter to reach the subjacent hepatic surface and to obtain hepatic dullness through the tympanitic superjacent colon. Similarly, in the presence of well established hypertrophic emphysema, the lower border of the right lung is extended and comes to overlie a greater area of the anterior and lateral hepatic surfaces, thus simulating a diminution in the volume of the liver. Extreme tympanites acts similarly in masking the true level of the inferior border of the area of hepatic flatness. Similarly all conditions which are attended by increase of general intraabdominal pressure, such as ascites, a large tumor of the intestine or an ovarian cyst, by forcing the liver upward into the vault of the diaphragm cause a pseudodiminution in the volume of the liver as outlined by percussion.

Diminution of the area of hepatic dullness attributable to diminution of the volume of the liver is encountered in acute yellow atrophy, atrophic hepatic cirrhosis, and hepatic atrophy consecutive to obliteration of the biliary passages. In the case of acute yellow atrophy there is produced during the course of a few days a very notable diminution in the volume of the liver, which is contracted into a small pulpy mass adjacent to the vertebral column and covered by coils of the small intestine. In the case of atrophic hepatic cirrhosis the retraction of the gland is first noted in the left lobe, which is retracted toward the right, with the production of a gastrocardiac limit of the semilunar space of Traube, which comes to intervene between the gastropulmonary and gastrohepatic limits of this area of frank gastric tympany. This form of hepatic cirrhosis is usually attended by enlargement of the spleen and ascites. In hepatic atrophy consecutive to obliteration of the biliary passages, the diminution in volume of the liver is preceded by a transient hypertrophy of the organ, and is accompanied by chronic icterus.

Increase of the area of hepatic dullness is encountered in the presence of hypertrophic cirrhosis and in degeneration of the organ, in the presence of hepatic tumor or abscess, and in simple congestion of the organ. From the clinical standpoint it is desirable to distinguish two varieties of hepatic hypertrophy; namely, hypertrophy in which the liver preserves its normal contour, and hypertrophy of the organ which develops irregularly, with the presence of abnormal protrusions upon the hepatic surface.

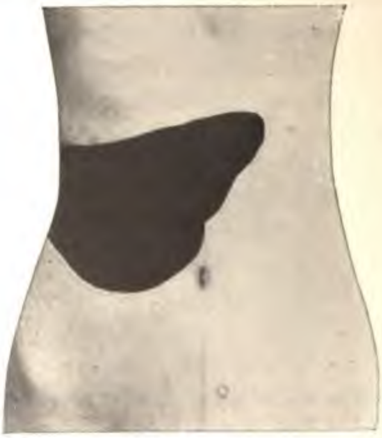


Fig. 251.—Indicating the region for dullness from enlarged liver. (From Crossen.)

A generally enlarged liver with maintenance of the normal contour is encountered in the hypertrophic cirrhosis of Hanot. A similar regular enlargement of the liver, but which is not frequently attended by jaundice, is encountered in fatty infiltration and chronic passive congestion of the liver in its earlier stages, in chronic malarial intoxication, during the acute infectious fevers, and in diabetes and amyloid disease of the liver, as well as in Banti's disease.

Irregular hypertrophy of the liver, with alteration of the no mal contour of the organ in the form of multiple nodules upon surface or a single extensive protuberance of the surface of the organ, is encountered in connection with carcinomatous infiltration of the liver, syphilis of the liver, hepatic abscess, or hydatid disease of the liver.

While increase in the volume of the liver results in extension of the areas of hepatic dullness and flatness, it is to be borne in mind that not every extension of these areas is symptomatic of hepatic enlargement. In all cases in which the inferior limit of the area of hepatic flatness is extended, the superior limit of hepatic dullness should be carefully delimited as well as the left sharp extremity of the left lobe of the organ above Traube's semilunar space. In true hepatic hypertrophy the area of hepatic dullness is extended in one or all of its dimensions. When the lower limit is established, the examiner should proceed to the delimitation of the superior limit with a view to the determination whether this limit occupies its normal level, whether it is elevated, or whether it is lowered, indicating in the last instance a depression of the organ rather than hypertrophy. In true enlargement of the liver the left extremity of the left lobe of the organ extends ordinarily well over into the left hypochondriac region, it may be to impinge against the spleen, with the consequent obliteration of the gastropulmonary limit of the semilunar space of Traube.

A pseudoextension of hepatic dullness superiorly is produced in the case of extensive effusion into the right pleural sac, in which event the continuous dullness may be confused with an extension of hepatic dullness upward, such as not infrequently attends hydatid cyst of the liver. The examiner should be circumspect in the differential diagnosis of these two states and should bear in mind the principle laid down by Frerichs; namely, that in the case of hydatid disease of the liver the superior limit of the zone of dullness presents its convexity superiorly, whereas in the event of pleurisy with effusion, the dullness presents an inferior convexity. A right-sided lobar pneumonia is quite as likely to cause confusion in the establishment of the superior limit of the hepatic dullness in any case in which hepatic enlargement under these circumstances is suspected. In the latter event the examiner should seek for bronchial respiration, râles, and increased vocal fremitus over the upper regions of the dull area.

A pseudoextension of hepatic dullness downward and toward the left may be encountered when the stomach and intestines contain solid material or when they are the seat of neoplasm. In the first instance evacuation of the gastrointestinal canal clears the confusing signs; while in the second case the examiner should resort to methodical palpation of the epigastrium and to auscultatory percussion of the various organs in the effort to differentiate the site of the growth, if any be present. Tumors of the stomach or intestine upon this examination are mingled with intestinal tympany and reveal amphoric phenomena, both of which are absent in the case of growths springing from the liver.

Finally, in any case in which it is a question of differentiation of collections situated above and below the diaphragm, free resort should be had to fluoroscopy in the differential diagnosis.

Displacement of the Liver.—As stated in a previous paragraph, instead of being increased in extent in one or more of its dimensions, the area of hepatic dullness may be displaced in its entirety. This displacement is usually manifested in a vertical direction.

Downward displacement of the liver may be occasioned by the pressure upon the superior aspect of the diaphragm by hypertrophic emphysema, pneumothorax, right-sided pleurisy with effusion, cardiac hypertrophy, or large pericardial effusion. Similarly, the liver may be depressed by a subphrenic abscess. Finally, the liver may participate in a general visceroptosis and occupy a lower level in the abdominal cavity than is normal. In the case of the "floating liver" of Cantani, occurring in multiparæ, the liver may occupy the right iliac fossa.

Downward displacement of the liver is differentiated from an increase in volume of the organ by demonstrating by percussion that the superior limit of hepatic dullness occupies a lower level than is normal.

Upward displacement of the liver occurs as a result of pressure exerted upon the inferior surface of the organ by ascites, tympanites, or increased abdominal tension due to tumor of an abdominal organ or ovarian cyst. Upward displacement may, however, be the sequence of diminished intrathoracic tension such as occurs with fibroid retraction of the right lung; or it may be the result of paralysis of the diaphragm. In upward displacement of the liver it may be demonstrated by percussion that both the superior and inferior limits of hepatic dullness are elevated and not the superior limit alone.

Auscultation.—Auscultation over the hepatic area occasionally elicits a friction sound i[illegible]tion with peritoneal involvement in the course of pe[illegible]sis, gallstone crepitations have been elicited upon auscultation over the fundus of the gall bladder.

Leopold, Martini, and Gabbi have described vascular murmurs upon auscultation of the liver in the presence of hepatic carcinoma and sarcoma, and in hepatic cirrhosis in which the blood vessels of the liver are constricted at several points in their course. Similar murmurs have been described in the presence of aneurysm of the hepatic artery.

Eustis has encountered bronchophony upon auscultation of the hepatic region in the presence of hepatic abscess which had perforated the diaphragm and established a bronchial communication.

CHAPTER XXVI

EXAMINATION OF THE SPLEEN, KIDNEYS, BLADDER AND URETERS

EXAMINATION OF THE SPLEEN

Clinical Anatomy.—The spleen is a solid organ situated deeply in the left hypochondriac region. The organ occupies an oblique position in the abdominal cavity, between the fundus of the stomach, the left kidney, and the diaphragm, its long axis corresponding to the course of the tenth rib. Owing to its accurate apposition with the inferior aspect of the diaphragm, the spleen exhibits respiratory mobility, and also a certain range of mobility with changes in the bodily posture.

While the normal spleen is subject to certain variations in form, its usual contour is elongated with a considerable degree of flattening, and when the spleen assumes this form it presents three surfaces, namely, a diaphragmatic surface, a gastric surface, and a less extensive renal surface. The diaphragmatic surface of the spleen is closely applied to the inferior surface of the left vault of the diaphragm, which separates the organ from the pleural cavity and the lower border of the left lung between the ninth, tenth, and eleventh ribs. The gastric surface of the spleen, directed forward, inward, and downward, is in relation with the fundus of the stomach. The renal surface of the spleen, directed inward and downward, is the least extensive of the three surfaces of the organ. It is in relation with the outer aspect of the superior pole of the left kidney and occasionally with a portion of the left suprarenal capsule.

The superior or posterior extremity of the spleen is on a level with the eleventh thoracic vertebra, and occupies a position one and one-half inches from the midspinal line. The inferior or anterior extremity of the organ reaches the midaxillary line, and occasionally the anterior axillary line in the normal subject.
The anterior extremity of the spleen commonly reaches as
forward as the costoarticular
upon the anterolateral wa
ity of the eleventh left
tion. Projection of the

this line is indicative of enlargement or displacement of the organ.

The spleen is maintained in its normal habitat through the medium of the splenic ligaments, composed of folds of peritoneum, and by the intraabdominal tension exercised upon its inferior surface, and counterbalanced by the intrathoracic tension exercised upon the superior aspect of the diaphragm.

The spleen is invested by a fibrous capsule and by a superjacent investment of peritoneum, save at the hilum of the organ situated upon the margin intervening between the gastric and the renal surfaces of the organ, at which point the splenic vessels enter and leave the organ. In the presence of inflammation of the organ the peritoneal coat frequently becomes roughened and rugose, with the production of the peritoneal friction sounds of Beatty-Bright. Moreover, as the splenic vein forms one of the principal tributaries of the portal vein, in the presence of valvular disease of the heart, or in portal obstruction from hepatic cirrhosis, the spleen becomes the seat of passive congestion with enlargement of the organ.

The size and weight of the spleen present numerous variations at different periods of life and under varying circumstances in the adult subject. The normal adult spleen is approximately five inches in length, three inches in breadth, and one inch in thickness. The spleen exhibits a moderate increase in volume during digestion; and the organ is regularly of ample dimensions in the well nourished subject, to become very small during starvation.

The weight of the spleen in proportion to that of the entire body is approximately the same at birth as it is in the case of the adult subject. At birth the proportion is 1:350, while in the healthy adult subject it is 1:320 to 1:400. With advancing age, however, the organ not only decreases considerably in weight, but even decreases considerably in proportion to the general weight of the body, frequently falling to the proportion of 1:700.

Occasionally in the healthy adult the spleen possesses a roughly tetrahedral form, while in other instances it is irregularly quadrilateral, with the result that instead of the long axis of the organ conforming to the course of the tenth rib, it occupies a plane perpendicular to this rib.

The spleen commonly corresponds to an area upon the surface of the body extending from the upper border of the ninth rib to the lower border of the eleventh rib, its inner extremity being situated one and one-half inches from the midspinal line, and its anterior extremity reaching as far forward as the midaxillary or the anterior axillary line. Superiorly the area of splenic dullness is limited by the inferior border of pulmonary resonance of the left lung, while inferiorly it is continuous with the area of renal dullness.

Physical Examination.—*Inspection.*—Inspection of the abdomen with the view of detecting variations in the size and position of the spleen is preferably practiced with the subject in the upright station or in the sitting posture, though it is occasionally desirable to employ other attitudes which will be mentioned in a subsequent paragraph. The normal spleen furnishes no evidence of its existence upon inspection of the abdominal surface. In the presence of enlargement of the organ, even, there is no evidence of the hypertrophy upon inspection in every instance. As a general rule the splenic enlargements attending the acute infectious fevers are not demonstrable upon inspection, owing partially to the fact that the splenic hypertrophy is only moderate in degree, and partially to the fact that the consistence of the organ is not materially influenced in these states, and while enlarged, it does not produce protrusion of the abdominal walls or flaring of the left costal arch. It is in chronic hypertrophies of the organ, in which the consistence of the organ is increased, that the examiner encounters fullness in the splenic region and flaring of the lower left costal arch. This protrusion of the abdominal wall is best recognized by the use of oblique illumination.

If the splenic enlargement is not too extensive, the organ presents two varieties of mobility; namely respiratory mobility coinciding with the movements of respiration, and postural mobility, depending upon changes in the attitude of the body for its induction. When the subject is placed in the right lateral decubitus, the tumor is displaced toward the right side of the abdomen, while in the left lateral decubitus it falls toward the opposite side of the abdominal cavity. Upon assuming the dorsal decubitus the splenic tumor ascends to a slight degree, to fall again upon assuming the erect posture. The respiratory movements of the spleen are never as extensive as are those of the liver, as the spleen is apposed to the inferior aspect of the diaphragm to a lesser degree than is the superior surface of the liver.

The examiner should make a distinction between acute enlargement of the spleen, which is transient and is not attended by appreciable alteration of the consistence of the organ, and is only rarely sufficiently pronounced to produce bulging of the abdominal wall, and chronic splenic enlargement, which is attended various degrees of hardening of the spleen, and which is constantly attended by physical manifestations of the splenic hypertrophy. Acute hypertrophy of the spleen attends many acute infectious diseases, notably diphtheria, scarlet fever, variola, pneumonia, influenza, acute miliary tuberculosis, typhoid and paratyphoid fevers, typhus fever, erysipelas, and septicemia. Chronic hypertrophy of the spleen is encountered in connection with chronic valvular disease of the heart, atrophic hepatic cirrhosis, splenomedullary leukemia, lymphatic leukemia, splenic anemia, chronic malarial intoxication, Hodgkin's disease, splenic infarction, and in amyloid and syphilitic disease of the spleen. In this category also come morbid growths of the spleen, such as carcinoma, sarcoma, and cystic disease of the organ. Under these circumstances the splenic enlargement is occasionally so extensive as to largely fill the abdominal cavity and cause confusion in differential diagnosis between the spleen and ovarian cyst.

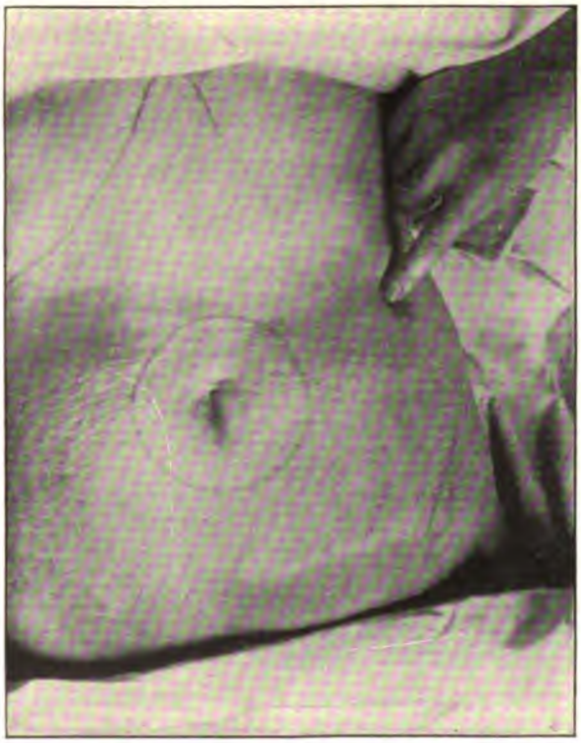


Fig. 252.—Indicating the area in which to search for splenic tenderness or enlargement. When the spleen is diseased it usually becomes enlarged and heavy and sinks below the margin of the ribs at the point indicated. (From Crossen.)

Palpation.—In the normal adult subject with normal abdominal walls and with the normal spleen occupying its normal habitat beneath the left vault of the diaphragm, palpation of the spleen is not possible, in whatever position the subject may be placed.

The examiner should endeavor in the first instance to palpate the spleen with the subject in the dorsal decubitus; after which it may become desirable to practice palpation of the organ in the right diagonal position of Schuster.

With the subject in the dorsal decubitus the examiner, seated by the left side of the patient, should apply the left hand, reinforced by the superimposed finger-tips of the right hand flat upon the abdomen in such position that the finger-tips of the left hand may be inserted beneath the left costal arch. If the spleen is enlarged, no difficulty will be experienced in palpating its lower border. The normal organ, however, as stated in a preceding paragraph, is not palpable. But the failure to encounter the inferior border of the organ by the above procedure shall not be interpreted as proof of the absence of splenic hypertrophy. If the spleen has not been encountered by simply sinking the finger-tips beneath the left costal arch, the subject to inspi deeply. Upon making

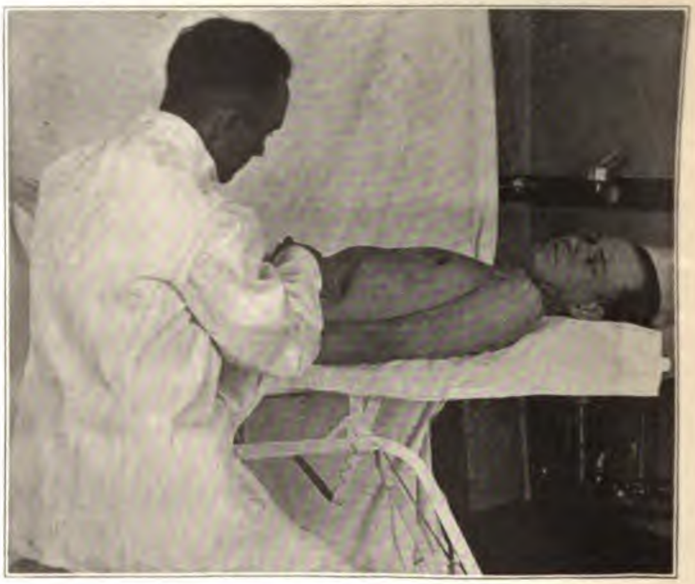


Fig. 253.—Palpation of the spleen.

spiration, the lower border of the organ, in the presence of moderate splenic hypertrophy, will be felt as it descends with the diaphragm during inspiration.

If the spleen is not encountered during this last maneuver, the examiner may resort to palpation in the right diagonal posture of Schuster. The subject assumes a recumbent posture midway between the dorsal decubitus and the right lateral decubitus, at the same time elevating the left arm and placing it behind the head. When the proper posture is attained, the patient reposes upon the right scapula. If now the finger-tips of the examiner are insinuated beneath the left costal arch during profound inspiration, the inferior border of the spleen will frequently become palpable as it descends.

Some difficulty may arise in determining whether a palpable tumor encountered below the left costal arch is of splenic or renal origin. In this connection it should be recalled that the spleen moves with respiration, whereas the kidney possesses no respiratory mobility. Moreover, as the kidney is overlapped by the large intestine, an enlargement of the kidney pushes the tympanitic gut before it, whereas the spleen occupies a position in front of the intestine. Finally, the shape of the two organs differs, the spleen being more or less oval, elongated, or tetrahedral with a sharp inferior border, while the kidney is smooth and reniform. Palpation is the method of choice in the exploration of variations in the contour, size, and position of the spleen.

During palpation of the spleen the examiner should take note of its form, dimensions, consistence, sensibility, mobility, the state of the surface, the presence of splenic friction fremitus or of pulsations, and the site in which the organ is encountered.

The hypertrophied spleen may conserve its normal form, or this contour may be markedly distorted from the normal, the contour of the organ in the two instances offering some indications as to the cause of the hypertrophy. The moderate acute enlargements of the organ are quite constantly attended by the preservation of the normal contour of the organ, as are also the chronic enlargements due to diseases of the blood or to chronic valvular disease of the heart. In the case of splenic enlargement due to malignant disease, to cystic disease, or to abscess of the gland, however, the normal contour of the organ is distorted and one or more protrusions may sometimes be palpated upon its surface.

The *dimensions* of the hypertrophied spleen present very numerous gradations from the moderate acute hypertrophies which are scarcely palpable to the immense leukemic spleen which may occupy the major portion of the abdominal cavity. Hyrtl encountered at autopsy an enlarged and indurated spleen which filled the abdominal cavity and which had created a pressure perforation of the left iliac bone.

The *consistence* of the enlarged spleen varies with the cause of the hypertrophy, the volume of the organ, and, with the duration of the enlargement. Acute transient hypertrophies of the spleen arising during the course of the acute infections are not attended by any notable alteration in the consistence of the spleen, and such hypertrophies are very frequently not palpable for this reason. In the immense hypertrophies of leukemia and splenic anemia, on the contrary, the organ is hard and occasionally of almost cartilaginous consistence. The amyloid spleen possesses a ligneous consistence in its advanced stages. Splenic tumors may present multiple hard masses, or a single large protrusion which may yield fluctuation. Barbieri has elicited fluctuation in the case of a large splenic abscess which was afterward treated surgically. Similarly, in the case of hydatid cyst of the spleen fluctuation may occasionally be demonstrated, as well as hydatid fremitus upon palpatory percussion.

Tenderness upon palpation of the spleen is as a rule only elicited upon the exertion of deep pressure, and in this case it is rather to be attributed to stretching of the capsule of the organ than to irritation of the splenic parenchyma. In perisplenitis attending acute splenitis or the enlarged spleen of acute splenic hyperemia tenderness is most readily elicited. Splenic tumors rarely exhibit tenderness with the exception of splenic carcinoma. *Signorelli's spleen point,* to which pain is referred in splenic disease is situated immediately below the junction of the fifth left costal cartilage and the midclavicular line.

Splenic enlargements exhibit rather a remarkable range of *mobility*. Like the liver, the spleen exhibits respiratory mobility, the enlarged organ descending with each inspiration, only to remount during expiration. In the upright attitude splenic enlargements occupy a lower position in the abdominal cavity than obtains during the dorsal decubitus. Upon assuming the right lateral decubitus, an enlarged spleen deviates toward the right side of the abdominal cavity and at the same time downward; while upon assuming the left lateral decubitus the opposite range of mobility is noted. The enlarged spleen is readily mobile upon manual manipulation.

A further element in splenic mobility consists in relaxation of the splenic ligaments, particularly in the case of multiparæ, whereby even in the case of a spleen of normal dimensions a condition of "floating spleen" is frequently created. Under these circumstances the organ may be encountered in the pelvis and Morgagni and Ruysch once found the viscus displaced into an inguinal hernia. The displaced spleen is ordinarily recognized by the palpable hilum of the organ, in which position the pulsations of the splenic vessels are occasionally detected. Moreover, the area of splenic dullness between the ninth and eleventh left ribs is replaced by intestinal tympany, only to be restored upon the manual reposition of the organ. A movable spleen may occupy a position superjacent to the abdominal aorta and have the pulsations of this vessel communicated to it in the form of a systolic rise and diastolic fall of the tumor. Placing the patient in the genupectoral posture causes the spleen to fall away from the vessel with the consequent suppression of the pseudopulsation.

The *surface* of the enlarged spleen may be smooth and uniform, or it may be irregular and embossed. The acutely hypertrophied spleen and the spleen of the earlier stages of chronic passive congestion present a smooth and uniform surface; while the spleen which is the site of infarction, abscess, echinococcus cyst, gummata, or of carcinomatous infiltration presents an irregular surface with nodules or bosses which may or not be umbilicated.

Friction fremitus is occasionally appreciated upon palpation of the abdomen over the spleen which is the seat of perisplenitis. This alteration of the splenic surface is much more frequently translated in the form of an audible friction sound, however, than it is demonstrable as a friction fremitus. Occasionally in the case of hydatid disease of the spleen one can elicit hydatid fremitus over the organ; but this sign is not present in the case of hydatid cyst of the spleen with the same frequency with which it is encountered over the lung or the liver.

Theoretically the systolic pulsation of the liver attending advanced tricuspid regurgitation should be appreciable over the spleen; but clinically it is the exception to encounter such a systolic pulsation. Pryor and Drasche have, however, described pulsations of the spleen in subjects suffering with aortic insufficiency.

Aside from the floating or wandering spleen which develops as a result of relaxation of the suspensory ligaments of the organ, the spleen may be displaced in a vertical direction by alterations

Owing to the intimate and continuous relation of the inferior border of the spleen with the superior pole of the left kidney internal to the left scapular line, it is impossible to establish by percussion the inferior limit of the spleen upon the posterior surface of the thorax. The superior limit of the organ is established by determining the inferior limit of pulmonary resonance in the scapular, postaxillary, and midaxillary lines; but here again, in the presence of effusion into the left pleural sac or in the presence of consolidation of the inferior lobe of the left lung it becomes impossible to establish the superior limit of splenic dullness and hypertrophy of the organ is apt to be suspected when it does not exist. A further source of error in splenic percussion is added when an enlargement of the liver causes the left extremity of the latter organ to invade the left hypochondriac region and to impinge against the spleen. In this event a false *spleno-hepatic angle* of tympany is established at the summit of Traube's semilunar space.

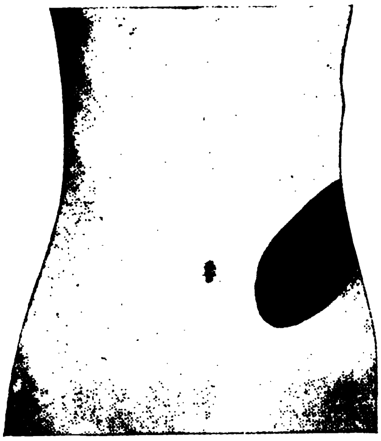


Fig. 254.—Indicating the region for dullness from enlarged spleen. (From Crossen.)

Percussion of the spleen has been practiced in all possible bodily attitudes in the hands of different clinicians. The subject has been placed in the abdominal position while the spleen was delimited upon the posterolateral aspect of the abdomen; he has been placed in the right lateral posture, which frequently causes the left iliac crest to impinge against the lower costal arch with obliteration of the left flank, which is a productive source of error; he has been placed in the right-lateral decubitus of Schuster; and Zeimssen has preferred to outline the spleen with the abdomen. As a rule, when the costoarticular line is reached, the dullness of the spleen will give place to gastric tympany in Traube's semilunar space. It is very rare for the anterior extremity of the normal spleen to surpass this line. Leichtenstern has objected to the employment of the costoarticular line in forming conclusions as to hypertrophy and displacement of the spleen, for the reason that the course of this line depends entirely upon the conformation of the thorax, with which the spleen has no direct connection. This author points out that in a subject with an elongated thorax it is quite possible for the anterior extremity of the spleen to surpass this line in the absence of hypertrophy of the organ; whereas in the case of deep chested subjects the anterior extremity of the hypertrophied organ may fall short of this line.

Percussion of the splenic region should be practiced during tranquil respiration as deep inspiration is capable of modifying the superior limit of splenic dullness markedly and the superior diminution in the area is not attended by a corresponding increase in the inferior limits of the dullness. This discrepancy occurs through the fact that the respiratory mobility of the spleen is limited on account of the relatively small extent of the organ which is apposed to the inferior diaphragmatic surface as compared to that of the liver; and while the inferior border of the lung descends into the complementary sinus of the pleura to the extent of one to one and one-half inches during full inspiration, the spleen is not displaced downward to a corresponding degree. Indeed, Gerhardt established the facts that while during full inspiration the inferior border of the left lung descended to the extent of three to four centimeters, the inspiratory displacement of the inferior splenic border did not exceed one centimeter.

In the pathologic state the examiner may encounter an increase in the area of splenic dullness, a decrease in the extent of this area, or a total absence of splenic dullness upon percussion.

The moderate hypertrophies of the spleen which accompany the acute infectious diseases are scarcely to be detected during percussion of the organ. But in the presence of extensive chronic hypertrophy of the organ the area may embrace a large portion of the abdominal surface. In more moderate enlargements of the spleen there is a coincident extension of the superior and inferior limits of the organ with extension of the anterior extremity of the organ toward the median line of the body. Thus, it not infrequently happens that the anterior extremity of the spleen comes into contact with the left lobe of the liver, with the production of a continuous band of dullness in the hypochondriac and epigastric regions, with the formation of a *splenohepatic angle* of gastric tympany at the summit of Traube's semilunar space. Auscultatory percussion is serviceable in separating enlargements of the spleen from growths springing from other abdominal organs; and examination of the blood frequently reveals corroborative findings.

Attention has been called in a previous paragraph to the inherent difficulties of delimitation of enlargements of the spleen by percussion and the possibility of effusions into the left pleural sac and the presence of solid accumulations in the stomach and the colon simulating extensions in the various limits of splenic dullness.

Diminution of the area of splenic dullness is encountered with the maximum frequency in connection with hypertrophic emphysema, when the voluminous lungs encroach upon the superior limits of the area of splenic dullness. Similarly, in the presence of tympanites the spleen is crowded into the left vault of the diaphragm and the area of splenic dullness is greatly restricted or abolished. In the aged subject the spleen frequently undergoes a striking diminution in size, with consequent restriction of the area of splenic dullness.

Absence of splenic dullness is noted in the case of the wandering spleen, in which the spleen has fallen from its normal habitat, and in the rarer cases of congenital absence of the organ. In the presence of pneumoperitoneum the collection of gas may insinuate itself between the spleen and the abdominal wall and temporarily abolish the dullness of the spleen.

Before basing conclusions as to the probable dimensions of the spleen upon the percussion findings, the student should pass in review the various factors which may influence these findings, and which have been enumerated in the foregoing paragraphs; and a diagnosis of splenic hypertrophy should not be formulated in any case in which the spleen does not become palpable.

Auscultation.—In the presence of perisplenitis a peritoneal friction sound is occasionally audible synchronous with the respiratory movements or provoked by pressure with the stethoscope. This friction sound is much more readily detected upon auscultation than is the accompanying fremitus upon palpation of the splenic region.

Occasionally during
ing leukemia, in the

of a floating spleen, vascular murmurs are detected upon auscultation of the splenic area. The significance of these vascular bruits have been variously interpreted by different observers and their precise mode of generation is still a subject of controversy. Gerhardt detected a double murmur over a pulsatile spleen in a subject of aortic insufficiency in connection with the double murmur of Duroziez over the femoral artery. Griesinger holds that vascular murmurs encountered over a hypertrophied spleen are due to the flow of blood through the large venous trunks; Tersti holds that they are due to twisting and relaxation of the afferent and efferent splenic vessels; Mosler attributes them to contractions of the splenic arteries; and Piazza believes that in the case of the indurated spleen they are due to constriction of the splenic arteries, while in the case of the soft spleen they are due to dilatation of the same vessels.

EXAMINATION OF THE KIDNEYS

Clinical Anatomy.—The kidneys are solid organs situated in the posterior portion of the abdomen, behind the peritoneum, upon either side of the vertebral column. The superior extremities of the kidneys correspond to the level of the upper border of the twelfth dorsal vertebra; while the inferior extremities are on a level with the third lumbar vertebra. Owing to its relation with the right lobe of the liver, the right kidney occupies a slightly lower level in the abdominal cavity than does the left kidney.

The normal adult kidney is approximately four and one-half inches in length, two and one-half inches in width, and two inches in thickness. The left kidney is slightly longer and narrower than is the right kidney. In the infant and the young child the kidneys are relatively larger than in the adult subject. The relative weight of the kidney in the adult to the entire body weight is 1:240, whereas in the infant the proportion is 1:120.

Posteriorly the kidneys rest upon the muscles of the posterior abdominal wall, the psoas, the quadratus lumborum, the fascia of the transversalis, and the diaphragm. The anterior surface of the right kidney is in relation with the inferior aspect of the liver, the descending portion of the duodenum and the hepatic flexure of the colon and the adrenal. The large intestine is united to the anterior face of the kidney by areolar tissue, with the result that in the presence of enlargement of the kidney from neoplasm or other cause the large intestine is carried before the kidney in its hypertrophy. The anterior surface of the left

The kidneys occupy portions of the epigastric, umbilical, hypochondriac, and lumbar regions upon either side of the median line. The superior pole extends as high in the epigastric region as a transverse line drawn about two inches below the ensiform process. The inferior pole extends below the subcostal line, only slightly in the case of the left kidney, and to a greater degree in the case of the right kidney. In the female subject the kidneys occupy a lower position in the abdominal cavity than in the male subject. In both sexes the superior pole of the organ is nearer the median line of the body than is the inferior pole.

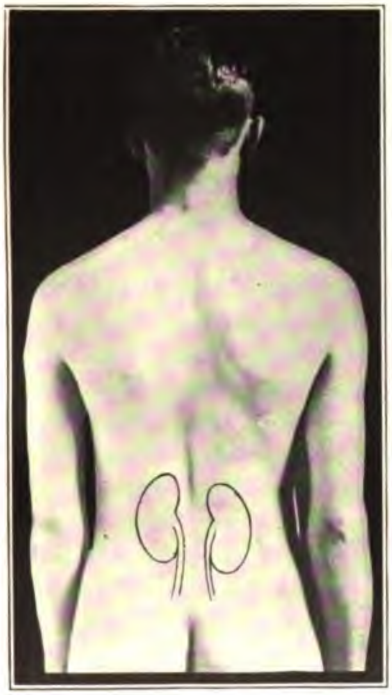


Fig. 257-*A*.—Topographic anatomy of kidneys and ureters.

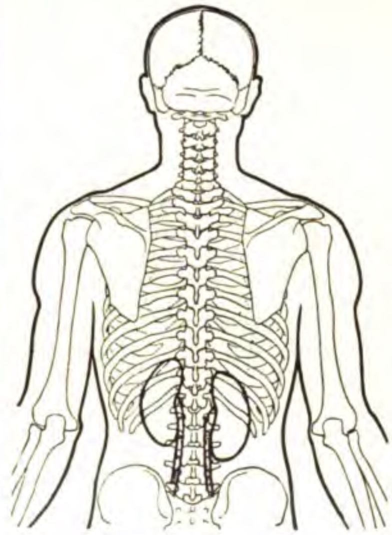


Fig. 257-*B*.—Topographic anatomy of kidneys and ureters.

The kidney is roughly reniform in shape, presenting a convex external border which conforms anatomically to the outer border of the quadratus lumborum muscle, and presenting a concave internal border, which bears in its midportion a deep fissure, the hilum of the kidney, for the entrance and exit of the renal vessels, nerves, lymphatics, and the ureters.

The kidney is maintained in position by a surrounding bed of adipose tissue, the perirenal fat, and by a sheath of fibrous tissue, which is continuous in the median line of the abdominal cavity and which is anchored superiorly with the fascia of the diaphragm.

Congenital absence of one kidney has been observed, and not infrequently one kidney is considerably larger than its fellow of the opposite side. Occasionally also the kidneys are fused at their inferior poles with the production of a "horseshoe kidney." In other instances the fusion of the organs is complete, the kidneys being represented by a single large organ with two ureters.

Physical Examination.—During physical examination of the kidneys inspection and palpation are the principal methods employed, the latter mode of examination in the majority of cases yielding much more definite and precise data than the former. In certain cases an attempt should be made to outline the kidneys by percussion, the technic and the difficulties of which are detailed in a subsequent paragraph. Auscultation is scarcely ever resorted to in the examination of the kidneys.

Inspection.—The normal kidney, occupying its normal situation, yields no evidence of its existence upon inspection of the abdomen. It is only in the presence of hypertrophy of the organ as a result of the development of renal neoplasms, extensive hydronephrosis, in the case of the large cystic kidney, or in the case of the displaced kidney that inspection of the abdomen reveals any abnormality in contour which is of localizing value.

A progressive enlargement of the kidney, whether it be due to neoplasm or other cause, produces moderate bulging of the abdominal wall in the first place limited to the lumbar regions of the abdomen, between the lower border of the costal arch and the iliac crest. In the further evolution of the hypertrophy the bulging progressively involves the umbilical region, producing ventral protrusion of the abdominal wall in this locality; and in the further growth of the organ, the liver or the spleen is crowded upward with the production of a variable degree of flaring of the costal arch upon the side of the enlarged kidney. Under these circumstances a renal enlargement is distinguished from a hypertrophy of the liver or of the spleen by the absence of respiratory excursions in the case of renal growths. In testing for respiratory excursion of the tumor, however, considerable care must be exercised in order to avoid confounding the movement of the abdominal wall over surface of the growth for a true respiratory enlargement, and it may be necessary to employ palpation in order to differentiate the two movements.

In the presence of extensive hypertrophy of the right or the left kidney, associated with bulging of the abdominal wall, there is occasionally observed a ridge traversing the prominent area of the abdominal wall, which corresponds to the course of the ascending colon in the case of the right kidney and to the course of the descending colon in the case of an enlargement of the left kidney. As the ascending colon is moored to the anterior surface of the right kidney by areolar tissue, when the kidney enlarges the colon is carried forward in front of the growth; and if the intestine contains gas, an elevation corresponding to the course of the ascending colon is produced upon the anterior abdominal wall. In the case of the right kidney this elevation pursues a course from below upward and toward the left side of the abdomen. In the case of enlargements of the left kidney this sign is frequently lacking, as the descending colon possesses no definite connection with the left kidney and frequently the kidney passes forward to meet the anterior abdominal wall within the limits of the descending colon.

The development of a perinephritic abscess in the perirenal tissues produces local bulging upon the posterior surface of the abdomen in the costovertebral angle occupying the interval between the twelfth rib and the vertebral column. When rupture of the abscess becomes imminent there is a circumscribed elevation of edematous or discolored integument at the site of imminent rupture. When these signs are in evidence, the examiner should practice very careful palpation of the vertebræ in the attempt to exclude a possible confusion with an abscess, the result of vertebral caries.

A displaced kidney is occasionally capable of producing a circumscribed prominence of the anterior abdominal wall, which may be situated low down in the abdominal cavity and may simulate a tumor of the ovary or other pelvic or abdominal organ. Bartels encountered a displaced kidney at the level of the right iliac fossa in a multipara with thin abdominal walls in which case it was possible to recognize the kidney by its characteristic reniform shape.

Palpation.—In the practice of palpation of the kidney the patient assumes the dorsal decubitus with the knees drawn up and supported, and the arms lying loosely at the sides.

The examiner sits upon the side of the kidney to be palpated and bimanual palpation is employed. The examiner exerts pressure with the index and middle fingers of the left hand in the women, particularly in subjects with moderate enteroptosis; with less frequency is the kidney movable in the male subject.

The normal kidney occupying its normal situation in the abdominal cavity is always palpated with difficulty and frequently is not palpable. But a movable kidney, a displaced kidney, and the kidney which is enlarged as a result of hydronephrosis, neoplasm, pyelonephrosis or surgical kidney is readily palpable and occasionally yields fluctuation under proper conditions.

When upon palpation of the kidneys the examiner encounters an abnormality in the dimensions or contour of the kidneys, he should proceed to make a detailed study of the sensibility, the form, and the volume of the kidney, its limits in respect to the adjacent abdominal viscera, its range of mobility, the state of its surface, and its consistence.

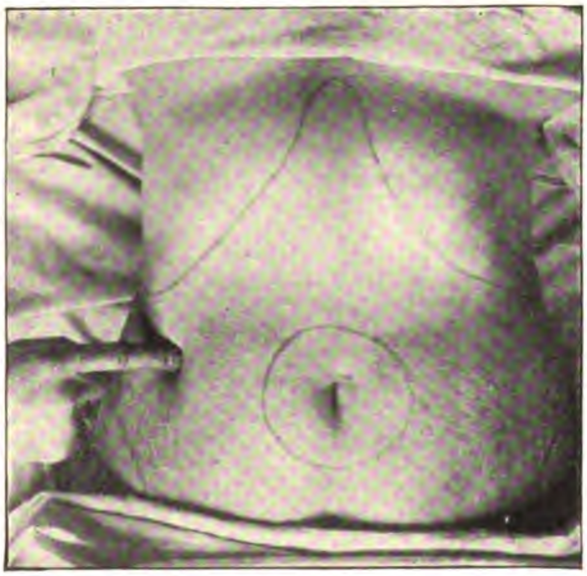


Fig. 259.—Indicating the region for kidney tenderness in front, on the right side. (From Crossen.)

The *sensibility* of the kidney is variable both in the normal subject and in pathologic states of the organ. The subjective sensation which is experienced upon compression of the normal organ is dull and aching, somewhat analogous to the sensation which is experienced upon moderate compression of the testicle. In the presence of pathologic changes in the organ the sensibility ranges from this dull pain to very lively, excruciating pain. Frerichs

ls attention to the fact that the left kidney is normally more
sitive to pressure than is the right kidney, probably as a result
its more superficial position, with the result that a greater degree
pressure is exerted upon this kidney during the routine palpa-
n of the kidneys. In renal disease tenderness may be elicited

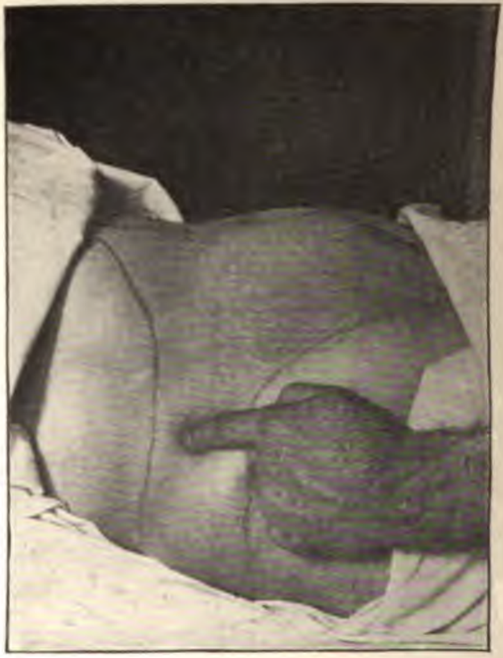


Fig. 260.—The point for kidney tenderness laterally. (From Crossen.)

—The point for ki m Crossen.)

upon pressure with the finger-tips upon the anterior abdominal wall two inches below the costal arch and slightly external to the mid-Poupart line. Upon the lateral wall of the abdomen tenderness is elicited upon pressure exerted at the same level. Posteriorly renal tenderness is elicited upon exerting pressure in the costovertebral angle.

The *form* of the kidney, even in pronounced enlargements of the organ is usually maintained to such a degree that the organ may be recognized by its reniform contour. Frequently it is considerably elongated; and in the case of the polycystic kidney is studded with regularly spherical nodules.

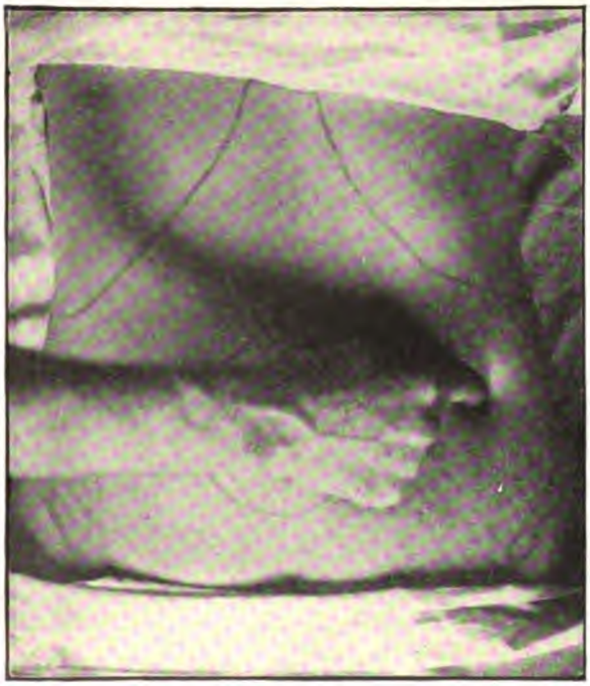


Fig. 262.—The area for left kidney tenderness in front. (From Crossen.)

The *volume* of the kidney in the presence of pathologic changes of the organ presents all gradations from the very moderate enlargement of the gland which scarcely renders it palpable to immense hypertrophy which fills a considerable portion of the abdominal cavity.

The examiner not infrequently encounters difficulty in the case of the pathologic kidney in establishing its limits in respect to the cystic disease of the organ; while those with irregular, nodular surfaces point to solid tumors of the organ, notably to carcinoma and sarcoma, and to the polycystic kidney. In cases of renal hypertrophy in which the colon is carried forward in front of the organ and is compressed between its anterior face and the abdominal wall the intestine is occasionally palpable in the form of a rounded band crossing the anterior surface of the kidney.

The *consistence* of the kidney in the presence of renal hypertrophy is frequently of considerable aid in the determination of the nature of the underlying lesion. In the case of solid tumors of the organ the tumor is hard and unyielding upon palpation. In the case of hydronephrosis, on the contrary, the organ is soft and yielding, and fluctuation is occasionally to be detected. Fluctuation is elicited most readily when the walls of the organ are not unduly tense; and an increase in the quantity of the fluid and of the tension of the walls renders the fluctuation less distinct. In the presence of echinococcus disease of the kidney it is occasionally possible to elicit hydatid fremitus over the site of the cyst.

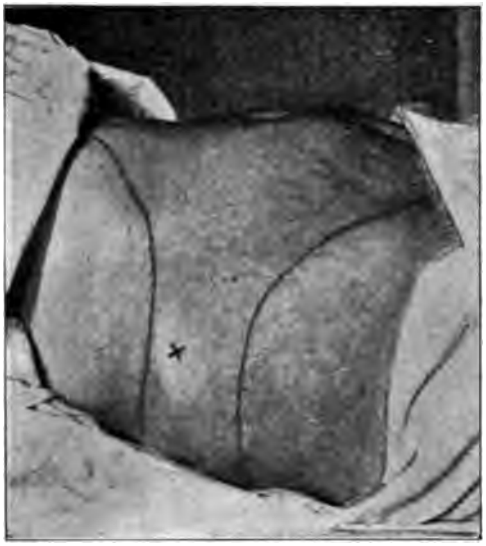


Fig. 264.—Point for kidney tenderness laterally. (From Crossen.)

Occasionally the examiner may be confused by a solid mass which crosses the median line low down in the abdominal cavity and which is continuous with the inferior poles of the kidneys. In this connection he should think of the possibility of the existence of the of the right kidney so that a renal hypertrophy carries the tympanitic colon before it, while tumors of the gall bladder, pylorus, or pancreas reach the abdominal wall nearer the median line, without the intervention of the tympanitic colon.

In the case of the left kidney, however, owing to the fact that there is no anatomic attachment of the kidney to the descending colon, the colon is frequently displaced, allowing the renal tumor to come forward and meet the abdominal wall. However, in these circumstances, the displaced colon will yield a tympanitic note along the border of the solid mass. Tumors of the spleen reach the abdominal wall above the transverse colon, which is displaced downward.

Hepatic enlargements exhibit distinct respiratory mobility, which is absent in the case of renal tumors; hepatic enlargements develop above the transverse colon, which they displace downward; and, as Bright showed, in the case of renal enlargements it is possible to insinuate the palpating hand between the growth and the anterior abdominal wall, while in the case of hepatic growths this maneuver is impossible.

Percussion.—In the physical examination of the kidneys, percussion is inferior to palpation as a method of examination, and its employment with satisfactory results is practically confined to the study of enlargements of the organs. In the case of the normal kidney it is impossible to map out any area of renal dullness upon the anterior surface of the abdomen, and it is exceedingly difficult in the case of the normal organs to establish any area of renal dullness upon the posterior abdominal wall which bears any definite anatomic relation to the true position of the kidneys. In the case of the normal kidneys, the superior limits of the organs are always obscured by their relations with the liver and with the spleen. It is equally impossible to delimit by percussion the internal concave borders of the organs on account of the thick musculature occupying the areas upon either side of the vertebral column and because of the fact that the transverse processes of the vertebræ frequently intervene between the internal borders of the organs and the posterior abdominal wall.

Externally it is possible, when the colon does not contain solid fecal accumulations to establish along the external border of the thick bed of muscles paralleling the vertebral column a limit where the so-called renal dullness gives place to intestinal tympany. This limit is commonly accepted as the external limit of renal dullness, and is assumed to represent the external border of the normal

kidney; but Weil established the fact that this lin
merely to the free edge of the massive muscular c
upon either side of the vertebral column and in no w
the anatomic position of the external border of the n
For the same anatomic reason the examiner will very r
in delimiting the inferior pole of the kidney, as it is co
thick superjacent musculature, which effectually deade
panitic sound which might be generated in the intes
level of the inferior pole of the kidney. Thus, percuss
kidney is practically limited to the delimitation of rena
ments, in which the hypertrophied organ approaches th
abdominal wall, thereby enabling the examiner to base hi
sions upon certain regional variations in the percussion
elicited upon this abdominal surface.

Here renal growths are differentiated from hepatic hy
phies by the absence of respiratory mobility, by the fact o
carrying the tympanitic colon in advance of them, and
impossibility in the case of hepatic growths of insinuati
finger-tips between the growth and the ventral abdominal

Similarly, growths springing from the spleen exhibit respi
mobility; they are situated higher up in the left hypocho
region than are renal growths; while the finding of the co
front of the growth pleads in favor of a renal growth, as gr
of the spleen develop above the level of the colon, which the
place downward.

Renal growths of extensive dimensions may be mistake
growths of the ovary. In this connection the examiner shou
call that renal growths develop from above downward, while tu
of the ovary develop from below upward; that renal tumor
ordinarily encountered behind the intestines and carry the tym
itic colon forward before them, while ovarian tumors are sit
in front of the intestine; and that renal tumors are attende
urinary changes, while ovarian growths provoke menstrual
turbances and uterine displacements.

Auscultation.—Auscultation is scarcely employed in the ex
nation of the kidneys. Bristone and Ballard have encount
vascular murmurs over the kidney in the presence of renal
cinoma, which simulated aneurysm of the abdominal aorta.

EXAMINATION OF THE BLADDER

The bladder is a hollow viscus, lying posterior to the sy
sis pubis. A pelvic organ in the adult s

bladder is situated in the abdominal cavity above the symphysis pubis.

Physical Examination.—*Inspection.*—The normal bladder causes no visible prominence of the abdominal surface; but when distended it produces bulging in the hypogastric region which, in extreme cases, may extend into the umbilical region. The cause of such distention may be prostatic hypertrophy, a lumbar cord lesion, or the comatose state of an acute infection.

Palpation.—The moderately distended bladder cannot be palpated through the abdominal wall; when, however, distended fully, it may be felt as a tense spherical mass in the hypogastric region.

Percussion.—Percussion is only available in cases of extreme distention of the bladder, when a flat note is elicited over the distended bladder in the hypogastric and umbilical regions, surrounded by a zone of intestinal tympany.

Auscultation is not employed in the physical examination of the bladder.

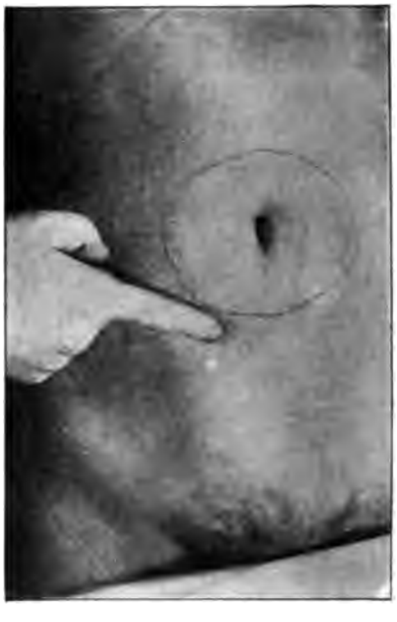


Fig. 266.—Indicating the site to search for tenderness of the right ureter. This may be found anywhere from the point indicated to some distance inside the circle, towards the umbilicus. (From Crossen.)

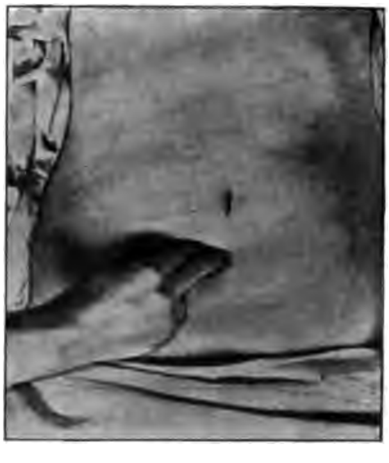


Fig. 267.—Palpating for tenderness or thickening about the right ureter. (From Crossen.)

EXAMINATION OF THE URETERS

The ureter is a cylindrical membranous tube, approximately from ten to twelve inches in length and one-sixth inch in diameter,

PART III. THE HEAD, NECK AND EXTREMITIES

SECTION I

THE HEAD AND NECK

CHAPTER XXVII

EXAMINATION OF THE HEAD

In the examination of the head the following points should be noted by the examiner:

1. Size and shape.
2. Condition of the fontanelles and sutures (in children).
3. Condition of the bones.
4. Condition of the hair.
5. Position of the head.
6. Movements of the head.

Size and Shape.—The head may be abnormally small (microcephalia) with premature closure of the fontanelles and sutures, a condition usually associated with idiocy.

A very large head is encountered in hydrocephalus, while moderate enlargement occurs in connection with rickets, cretinism and hypertrophia cerebri.

In *rickets* the circumference of the head is increased two or three inches, the enlargement being chiefly due to thickening of the cranial bones. The shape of the head is rather square (box head), owing to the presence of osteoid bosses upon the frontal and parietal regions. The rachitic head is flattened at the vertex and over the occiput. Soft, compressible areas, *craneotabes,* are often present; the fontanelles are widely open and the sutures are tardy in closing.

Concomitant signs of rickets are the rosary, the chicken or pigeon breast, spinal curvature, tumid belly, and changes in the extremities of the long bones.

Hydrocephalic Head.—In hydrocephalus the head is the circumference sometimes reaching 32 inches at t month of life. The large prominent forehead is in ma trast with the small face. The fontanelles and sutures a open, and the veins of the scalp are prominent and c The skull is very thin and may be translucent to can The child has difficulty in holding the head up.

While the head in hydrocephalus somewhat resemble chitic head, there are differences. In hydrocephalus the globular rather than square as in rickets; also in hydro the sutures and fontanelles are wider and the fontanell which is not true of rickets.

In *cretinism* the head is large, flattened at the vertex, w sutures and fontanelles, but without bulging. The facial e is dull, the nose flat, and the face large, with puffy eyeli extremities are short and thick; and the tongue is larg protruding from the mouth. There are pads of fat in th clavicular regions.

Fontanelles and Sutures.—The posterior fontanelle n closes about the end of the second month, while the anter tanelle closes between the eighteenth and twentieth mo life.

Tardy closure of the fontanelles occurs most frequently nection with rickets. Closure is also delayed in hydroc and cretinism. In rickets the fontanelles may remain open the fourth year of life.

Bulging fontanelles indicate increased intracranial pressu are noted in hydrocephalus, cerebral hemorrhage, meningiti tumor, sinus thrombosis, meningeal hemorrhage, and durin fevers.

Depressed fontanelles are noted during chronic wasting d in pulmonary diseases attended by dyspnea, after severe di during the early stages of meningitis, and in cholera infantu

Enlargement of the fontanelles, the anterior fontanelle ex one inch in diameter, is suggestive of rickets, hydrocephal tinism, and may be a hereditary condition.

Open Sutures.—The sutures of the child's head normall between the sixth and eighth months. Open sutures aft time are significant of rickets, cretinism, or hydrocephalus

Condition of the Bones of the Head.—A number of cha the bones of the head possess diagnost[illegible] [illegible]ificance.

Craniotabes, the presence of thin, compressible areas in the cranial bones, is symptomatic of rickets, infantile syphilis, or chondrodystrophy.

Osteoid bosses on the frontal and parietal bones in infants are symptomatic of rickets.

Soft, nodular swellings on the skull, which become harder with advancing age, are symptomatic of syphilitic periostitis of the cranial bones.

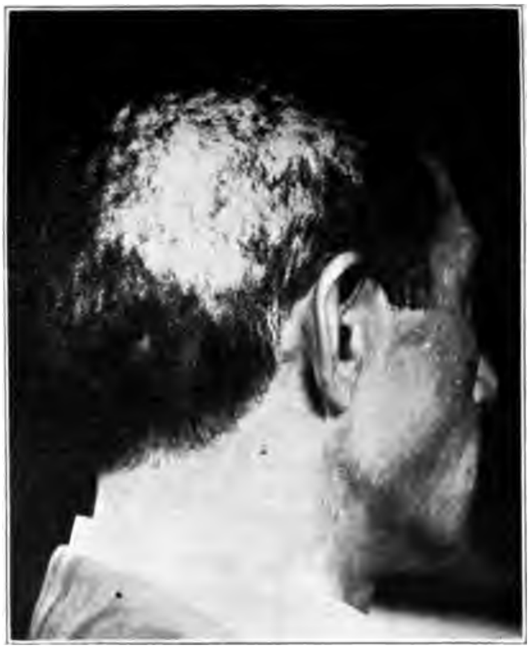


Fig. 268.—Alopecia areata. Numerous small patches have coalesced, forming a rather unusual picture, inasmuch as the baldness is not as complete as usual. (From Hazen.)

Tenderness over the mastoid process, with fever, deep-seated pain, and, if pus has formed, fluctuation, is symptomatic of inflammatory disease of the mastoid cells.

The Condition of the Hair.—*General falling* of the hair follows many acute febrile conditions, notably typhoid fever. General loss of hair also occurs in gout and myxedema.

Circumscribed falling of the hair, producing local areas of baldness, results from tinea tonsurans, scarring from local trauma neuralgia of the trigeminal nerve, and syphilis.

young children lateral deviation of the head may be due to injury to the muscles of the neck during parturition.

Abnormal fixation of the head is observed in connection with retropharyngeal abscess, cervical adenitis, rheumatism, arthritis deformans, extensive scars from burns, and in Pott's disease.

Movements of the Head.—*Nodding spasm,* a rhythmical up and down movement of the head, is observed in patients suffering with hysteria, and occasionally in connection with rickets. The movement may be continuous; or may be absent during quiescence and only brought out by excitement.

Arrhythmic, purposeless movements of the head occur in Sydenham's chorea.

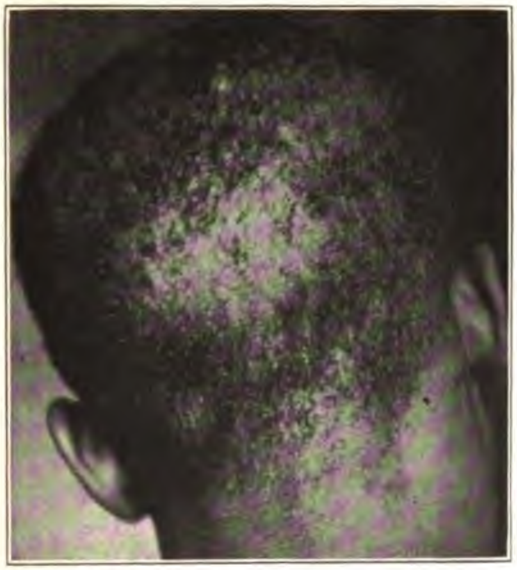


Fig. 270.—Syphilitic alopecia. (From Hazen.)

Spasmodic movements of the head, in which the head deviates laterally, occur in spasmodic torticollis.

Inability to move the head occurs in connection with the flaccid paralysis of acute anterior poliomyelitis, in caries of the cervical vertebræ, and in the late stages of cerebrospinal meningitis, and during comatose states from any cause.

The Ear.—Congenital Defects.—Among the congenital defects of the auricle may be mentioned entire absence of this portion of the auditory apparatus; excessive development or defective velopment of the auricle, macrotia and microtia respectively presence of more than one auricle, or polyotia; malformatio absence of the lobule, helix, or antihelix.

Fistula auris congenita, a rare defect, consists of a short b canal, lined with epithelium, with its orifice either in front or below the tragus.

Hematoma auris, or *othematoma*, is a bluish-red swelling invo ing the concha and fossa of the antihelix and helix, the lobule e caping. It is a trophoneurosis, and the condition is observed mo commonly among insane patients, in whom it was formerly at tributed to ill-treatment. A similar bluish discoloration, involving the entire auricle may follow trauma, due to effusion of blood beneath the perichondrium.

Tophi, small, hard nodules of sodium urate, are frequently found in the helix in gouty patients.

Cysts of the auricle, small, noninflammatory tumors, containing clear fluid, are sometimes encountered about the auricle. They are differentiated from perichondritis by the absence of pain and other inflammatory signs.

Sebaceous cysts, due to blocking of the ducts of the sebaceous glands and accumulation of the secretion, produce roundish tumors situated usually in the skin behind the lobule or in the lobule.

Blueness of the auricle occurs as a sign of cyanosis, and also in the early stages of frostbite, in which it becomes later yellowish-white.

Keloid may be encountered on the lobule, due to piercing the lobule for earrings, most commonly in the negro race.

Otomycosis.—In otomycosis, due to the growth of the Asper- illus Niger in the external auditory canal, the canal is studded ith black spots, which under the microscope reveal the presence f the fungus.

Discharge of blood from the external auditory meatus is in- ative of fracture of the base of the skull or otitis media. In case of fracture of the base of the skull the blood is mixed with ebrospinal fluid, which prevents coagulation; while in otitis lia there is admixture with pus. Discharge of pus unmixed blood indicates purulent otitis media or abscess.

CHAPTER XXVIII

EXAMINATION OF THE FACE

CONTOUR OF THE FACE

The contour of the face is altered by many diseases, chief among which may be mentioned acromegaly, hydrocephalus, osteitis deformans, leontiasis ossium, leprosy, and facial hemiatrophy and hemihypertrophy.

Acromegaly.—In acromegaly the face assumes an oval or elliptical shape, due to the enlargement of the frontal and malar bones, and the mandible, which become massive. Owing to this growth the teeth are separated by intervals, the lower teeth projecting beyond those of the upper jaw. The ears are large, the nose thickened, and the superciliary ridges are prominent. The tongue is large, sometimes protruding from the mouth. The eyes are unchanged; and, by contrast with the massive features, appear abnormally small.

Cretinism.—In cretinism the face is broad and flat, presenting a bloated appearance. The eyes are wide apart, the eyelids are thickened, and the nose is broad and negroid. There is pouting of the lips and protruding tongue, the child presenting a picture of imbecility.

Myxedema.—In myxedema the lines of expression in the face are obliterated by swelling in the subcutaneous tissue. The contour of the face has been likened to a "full moon." The nostrils and lips are large and thick, the mouth is enlarged, and there is usually a reddish patch over the cheek. Other signs of myxedema are the dry rough skin, the increase in bulk of the whole body, the inelastic swelling of the subcutaneous tissue, which does not pit upon pressure, and local deposits of subcutaneous tissue in the supraclavicular fossæ.

Hydrocephalus.—The face in this disease is triangular with the base of the triangle above. The features, which are of size, present a marked contrast with the enormous forehea

Osteitis Deformans.—The face in this disease is tria

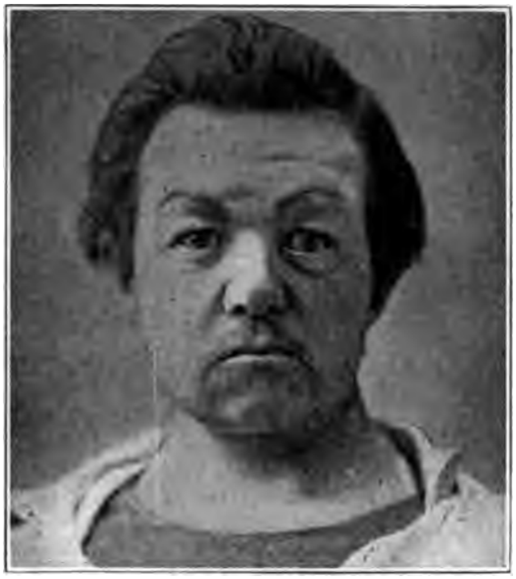


Fig. 273.—Face of myxedema. (Butler, after Gardinier.)

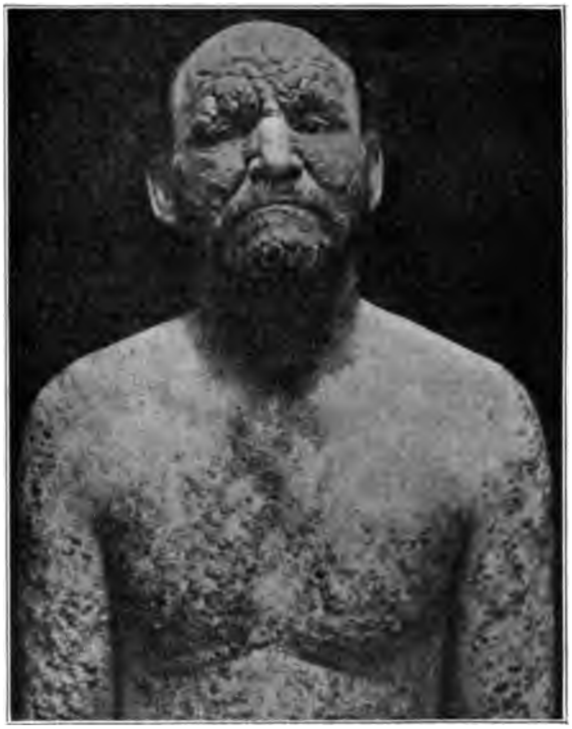


Fig. 274.—Leprosy. (From McFarland.)

with the base directed upward, owing to the thickening of the bones of the cranium. The head is carried in a position of forward inclination. The disease is associated with bowing of the bones of the upper and lower extremities, kyphosis, and not infrequently ankylosis of the spine.

Leontiasis Ossium.—This disease is characterized by progressive enlargement of the bones of the cranium and face, beginning usually in the superior maxillary bones. Blindness occasionally develops from pressure upon the optic nerves.

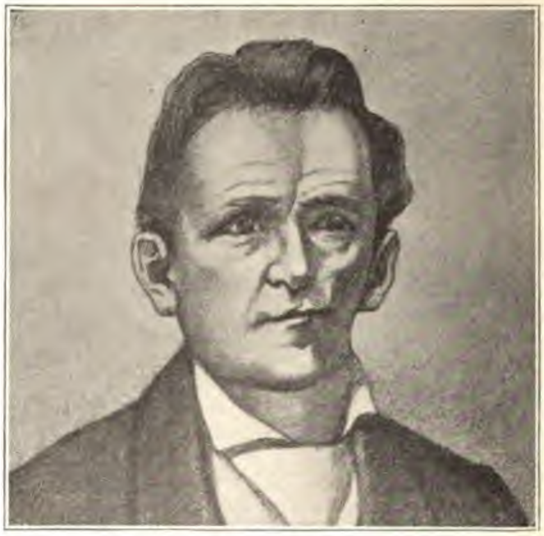


Fig. 275.—Facial hemiatrophy. (From Butler.)

Leprosy.—When the nodes of leprosy develop in the face they produce thickening of the skin of the forehead and cheeks. The nose is flat and thick; the lips are thick; the ears are thick and large, while the eyebrows, eyelashes, and beard are shed, constituting the Facies Leontina.

Facial Hemiatrophy.—In facial hemiatrophy one-half of the face is smaller than the opposite half, with a sharply defined vertical line of junction. The condition usually begins during childhood in one or two spots on one side of the face. The skin begins to undergo atrophic changes, followed by a similar involvement of the underlying subcutaneous tissue, muscles and bones.

The skin of the affected half of the face becomes wrinkled, the teeth become loose, the eyebrows fall out. The secretion of the sebaceous glands is diminished or abolished. The face is drawn toward the sound side, rendering the contrast between the two sides striking.

A similar facial asymmetry is encountered in children as a developmental defect, often in association with congenital torticollis.

Facial hemihypertrophy, the opposite condition, in which one side of the face is enlarged, occurs as an anomaly in the development of the face, sometimes associated with hemihypertrophy of the entire half of the body.

THE COLOR OF THE FACE

Pallor of the face occurs in anemia, ischemia, the edema of Bright's disease, and transiently as the result of sudden fright.

Flushing of the face may be transient and due to vasomotor disturbance, or may be persistent, notably in the early stage of acute fevers, as yellow fever. The flushed cheek of pneumonia and the bilateral flushing of tuberculosis have been described. A flushed face accompanies excessive cardiac hypertrophy, and in one form of essential anemia, namely, chlorosis rubra, is a marked feature of the disease. In apoplectic attacks and in the early stages of alcoholic intoxication the face is flushed.

Cyanosis, or bluish discoloration, particularly noticeable in the lips and ears, occurs in uncompensated heart disease. A similar bluish discoloration of the face is symptomatic of poisoning with coal tar products.

Yellowish discoloration of the entire face is suggestive of the cachexia of malignant disease, syphilis, or chronic malaria. A similar hue accompanies chronic constipation with inactive liver, certain cases of exophthalmic goiter and Addison's disease. A lemon yellow color of the face and body, with maintenance of the subcutaneous fat of the body, occurs with pernicious anemia.

Bluish discoloration, or argyria, occurs in cases of chronic silver poisoning.

Brownish, muddy patches upon the face, termed *chloasma*, frequently develops in pregnant women and in women with uterine or ovarian disease.

SPASM OF THE FACE

Spasm of the facial muscles occurs as a result of functional or organic disorders. It may be tonic or clonic, unilateral, or bilateral. It is more frequently encountered in women than in men. Among the conditions in which facial spasm possesses diagnostic significance may be mentioned:

Habit Spasm.—This spasm occurs in neurotic children, particularly in young girls. It is intensified by excitement or examination. It may consist in the rapid winking of an eye or the drawing up of one corner of the mouth. The neck muscles are frequently involved, the head being given a quick shake at the time of the winking.

Convulsive Tic.—This is a very sudden spasm of the facial muscles, frequently involving the brachial muscles as well. The spasmodic movements may be almost constant or may occur in paroxysms. In extreme cases the spasm may involve all the muscles of the body, the movements being very irregular and violent. The spasm is often accompanied by explosive utterances, echolalia and coprolalia.

Blepharospasm.—This is a sudden tonic contraction of the orbicularis palpebrarum muscle, causing partial or complete closure of the eye. More commonly the spasm affects the lateral facial muscles also, producing constant twitching of the side of the face. Usually unilateral, blepharospasm may be bilateral. The spasm is increased by emotional excitement and voluntary movement of the muscles of the face. If not reflex from irritation of the conjunctiva or cornea by a foreign body, it indicates involvement of the facial nerve.

Chorea.—Chorea produces arrhythmical jerking contractions of the facial muscles. It is accompanied by the other symptoms of the disease, as purposeless movements of the hands and feet.

Exophthalmic Goiter.—Spasm of the levator palpebræ superioris muscle, causing rapid movements of the upper lids, occurs occasionally in exophthalmic goiter, in which it constitutes Abadie's sign of this disease.

Tetanus.—Tetanus or lockjaw produces tonic spasm of the facial muscles, with the risus sardonicus, or sardonic smile which is characteristic of the disease.

Unilateral clonic spasm of one or more facial muscles points to irritation of the facial region of the cortex of the brain or to irritation of the facial nerve trunk in its course or at its exit by tumor or aneurysm of the vertebral artery.

THE FOREHEAD

The forehead should be examined for scars, skin eruptions, and nodular swellings.

Scars upon the forehead may be indicative of former traumatism or of the eruption of syphilis.

Eruptions.—The forehead is subject to many cutaneous eruptions, notably those of measles, smallpox, and syphilis, in which last named disease it constitutes the so-called corona veneris.

Nodular swellings of the forehead may be indicative of glanders, trichinosis, syphilitic periostitis, or tumor of the cranial bones.

THE EYES

The Eyelid

Edema.—Edema of the lids with puffiness, occurs in connection with the edema of nephritis and in anasarca due to cardiac disease or hepatic cirrhosis. Edema of the lids is also noticed during the active stage of pertussis, severe coryza, erysipelas, cerebral thrombosis, and in arsenic and iodine poisoning. Slight puffiness and edema of the lids upon arising in the morning is noted in certain persons as a normal phenomenon.

Duskiness of the lids and the infraorbital region is symptomatic of uterine and ovarian disease, pregnancy, anemia and exhausting disease, the molimina of menstruation, and it is said masturbation.

Xanthoma is a small, slightly elevated, flattened lipomatous new growth which is occasionally encountered on the eyelids of diabetic patients.

Ptosis.—Ptosis of the upper lid is usually due to syphilitic paralysis of the oculomotor nerve. Usually unilateral, ptosis may be bilateral. A bilateral ptosis of brief duration sometimes is seen in anemic and overworked women. Ptosis of the lid occurs with acute encephalitis and as a congenital condition.

Hordeolum.—A hordeolum or stye is a small abscess in the lid margin, situated at the root of an eyelash upon the anterior margin of the lid. Styes are acute, run a short course, but are prone to recur repeatedly.

Blepharitis Marginalis.—Blepharitis marginalis, inflammation of the lid margin is characterized by the formation of a series of scales or crusts along the lid margin, which upon removal expose a red, glazed surface. As the scales adhere to the lashes, they are sometimes mistaken for the eggs of pediculi.

Chalazion.—A chalazion or meibomian cyst is a small hard tumor of the upper eyelid, imbedded in the tarsal plate. It results from obstruction of a meibomian gland, and is prone to become inflamed and suppurate.

Epithelioma.—Epithelioma usually is seen upon the lower lid in persons past middle life. A history of long duration is usually obtainable.

Chancre.—The initial lesion of syphilis rarely occurs upon the lids.

Lagophthalmos.—Lagophthalmos, imperfect closure of the eyelids occurs with the exophthalmos of Graves' disease, and as a result of partial facial nerve paralysis.

The Conjunctiva

Pallor.—Pallor of the conjunctiva is a sign of anemia and calls for rather than replaces a blood examination.

Yellowness of the conjunctiva accompanies jaundice, and points to hepatic disorder.

Subconjunctival hemorrhage may occur during paroxysms of cough in pertussis or asthma, or as the result of local trauma.

Conjunctivitis.—In inflammation of the conjunctiva the membrane is red and bathed with mucopurulent or purulent discharge. Conjunctivitis may result from local infection, or may accompany the acute infectious diseases.

The Globe

Exophthalmos.—Exophthalmos, or protrusion of the globe of the eye, may be indicative of hemorrhage into the orbit, paralysis of the ocular muscles. [illegible] the superior longitudir sinus, tumor of [illegible] hi globe forward [illegible] unilateral or bilateral, the latter constituting one of the cardinal symptoms of exophthalmic goiter.

Von Graefe's sign of exophthalmic goiter consists in the inability of the upper lid margin to accurately follow the sclerocorneal junction downward during downward rotation of the globe of the eye.

Enophthalmos.—Recession of the globe of the eye into the orbit, enophthalmos, occurs in exhausting diseases, particularly those which are associated with the loss of tissue fluids, as cholera. Enophthalmos is also caused by absorption of the orbital adipose tissue during chronic wasting disease, notably in tuberculosis, diabetes, marasmus, and the cachexia of malignant disease.

Position of the Globe.—During epileptic seizures and hysterical coma the globes of the eyes rotate and turn upward. In hydrocephalus the globe looks downward, while following cranial injuries both globes look toward the side of the injury (conjugate deviation).

Oculocardiac Reflex.—Gentle pressure upon the eyeball of a normal subject produces a perceptible slowing of the pulse through vagus inhibition. This is a true reflex, the afferent impulse incited by pressure upon the globe of the eye being transmitted through the ophthalmic division of the trigeminal nerve to the Gasserian ganglion and thence through the larger root of the fifth cranial nerve to its root of origin. Thence the impulse is transmitted downward to the nucleus of origin of the vagus nerve, resulting in tonic efferent impulses from this center causing inhibition of the cardiac rate.

Abolition of this reflex indicates a break in the reflex arc at some point. Such abolition is noted in cerebrospinal syphilis and paresis. Abolition of this reflex is one of the earliest signs of syphilitic involvement of the central nervous system, and it is a sign which is readily elicited by the general practitioner. (Auer.)

Cornea and Sclera

Arcus Senilis.—The arcus senilis is a grayish line at the sclerocorneal junction which partially encircles the cornea. Present in many elderly persons the arcus senilis is particularly frequent in arteriosclerosis and chronic nephritis.

Interstitial Keratitis.—Inflammation of the interstitial tissue of the cornea, leading to partial opacity of this structure or to small pinkish "Salmon Patches" is nearly always a sign of hereditary syphilis. The condition is usually bilateral, affecting children between 5 and 15 years of age, girls being more frequently attacked than boys.

Ulceration.—Comparatively large ulcers of the cornea are apparent as losses of the surface epithelium; while minute ulcers may require the instillation into the eye of a few drops of Fluorescin, which stains the ulcer a bright yellow-green. Corneal ulceration frequently develops during the exposure of the cornea as a result of the exophthalmos of Graves' disease; and in cases in which the cornea is insensitive owing to disease of the ophthalmic division of the fifth cranial nerve. Corneal ulcer is prone to develop during prolonged fevers when the patient lies long with the eyes only partially closed.

Opacity.—Corneal opacity may result from the repair of a corneal ulcer, from interstitial keratitis, or as a result of Pannus. Corneal opacity sometimes develops during the course of scrofula or chronic malaria.

Staphyloma.—Staphyloma, a bulging of the cornea, usually is a sequence of weakening of the cornea by deep ulceration, particularly in connection with gonorrheal ophthalmia.

Yellow Sclerotics.—Yellow discoloration of the sclerotics occur in jaundice from hepatic disorder. Small, circumscribed, yellowish patches, pinguecula, are innocent growths springing from the ocular conjunctiva.

Bluish Sclerotics occur in connection with chlorosis, in which they contrast markedly with the greenish discoloration of the skin; also in nephritis, and Addison's disease.

Scleritis.—In inflammation of the sclerotic coat of the eye small bluish or purplish elevations are left upon the sclerotics.

THE NOSE

Shape.—The shape of the nose is altered by a growing tumor within the nasal cavities, or from the adjacent bones of the face. In cretinism and myxedema the nose is flattened and negroid. In syphilis in certain instances the nasal bridge is destroyed with the production of a characteristic deformity, the saddle nose.

Redness.—Redness of the nose, aside from being commonly associated with a history of chronic alcoholism, is observed in lupus erythematosus, in circulatory disturbances, chronic digestive disorders, and in amenorrhea.

Epistaxis.—Discharge of blood from the nose is frequently a sign of incipient typhoid fever. A discharge of blood mixed with cerebrospinal fluid occurs with fracture of the base of the skull. Discharge of blood from the nose may signify foreign bodies in the nose, acute catarrh, local hyperemia from cardiac disease, local ulceration which may be simple, carcinomatous, or syphilitic, or hemorrhagic diseases as hemophilia, scurvy, or purpura hemorrhagica.

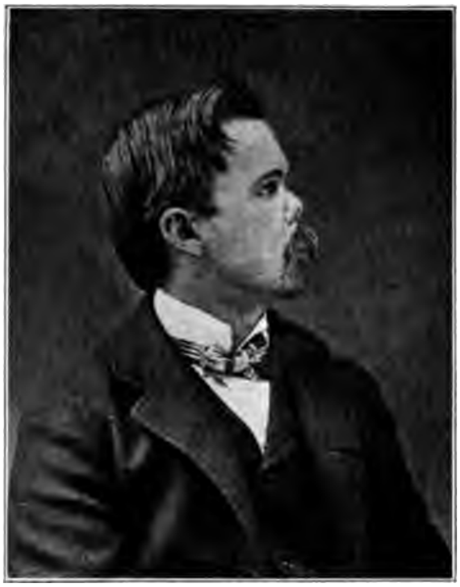


Fig. 276.—Saddle-nose. (From Eisendrath.)

Pseudomembrane.—A pseudomembrane develops in the nose in nasal diphtheria, a condition which is associated with considerable swelling of the associated lymph glands. The pseudomembrane may spread to the skin of the face, the conjunctiva, or the antrum of Highmore.

Adenoid Vegetations.—In the presence of adenoid vegetations in the nasopharynx the nose is but poorly developed, the nostrils appearing small and pinched.

Ulceration.—A chronic ulcer on the ala of the nose may be tuberculous, carcinomatous or syphilitic.

THE LIPS

Pallor of the lips suggests but does not prove the presence of anemia.

Cyanosis, or blueness of the lips, if not due to the ingestion of large doses of coal-tar products, is indicative of regurgitant heart disease or pulmonary disease of an obstructive nature as e physema and pneumonia.

Fig. 277.—Mucous patches. (From Hazen.)

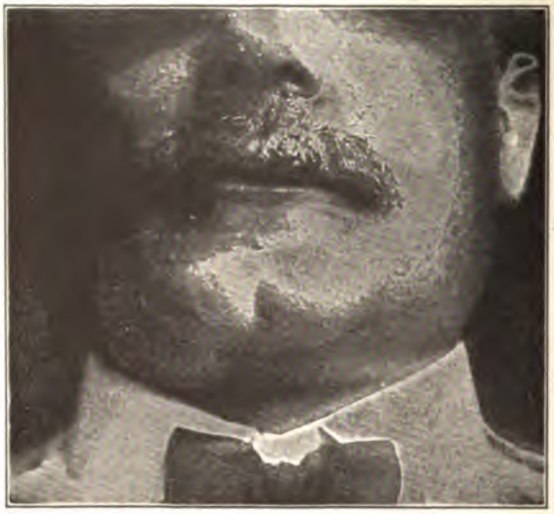


Fig. 278.—Chancre of the lip of one month's duration. (From Hazen.)

Parted lips, when dry and cyanotic indicate the dyspnea of cardiac or pulmonary disease. Parted lips in a child with a small, pinched nose is suggestive of adenoid vegetations in the nasopharynx.

Loose, pendulous lower lip accompanies chronic bulbar palsy, and less frequently is seen with diphtheritic palsy.

Herpes of the lips, herpes labialis, occurs with pneumonia most frequently, less frequently with malaria, and typhoid fever, and other febrile affections.

Enlargement of the lips accompanies angioneurotic edema, local abscess formation, and phlegmonous inflammation, and obstruction of the lymphatics draining the lips, *macrocheilia.*

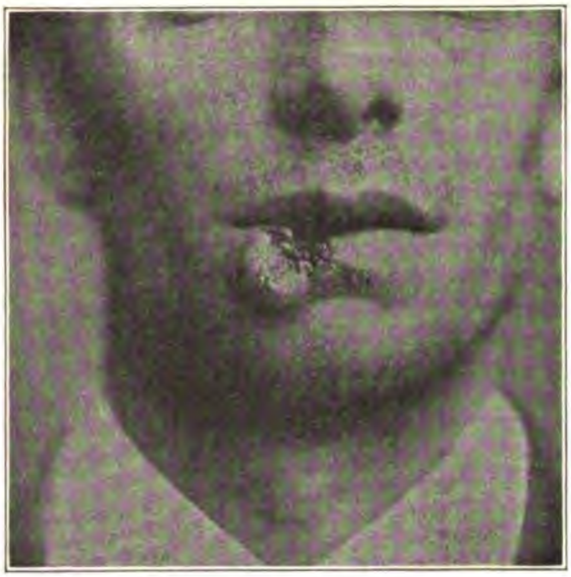


Fig. 279.—Prickle-celled carcinoma of the lower lip in a young man, which arose after treating a clinically benign lesion with caustic pastes. (Gilchrist's collection.) (From Hazen.)

Rhagades, or fissures of the lips, usually affect the lower lip near its center, in cold dry weather. Similar fissures developing upon the lips near the angle of the mouth in a child are good signs of hereditary syphilis.

Mucous Patch.—Flat, whitish sores near the angles of the lips with sharply defined borders, are mucous patches of syphilis.

Chancre.—An indurated sore on the lip, particularly when developing in a young person, and associated with enlargement of the associated lymph glands suggests the initial lesion of syphilis.

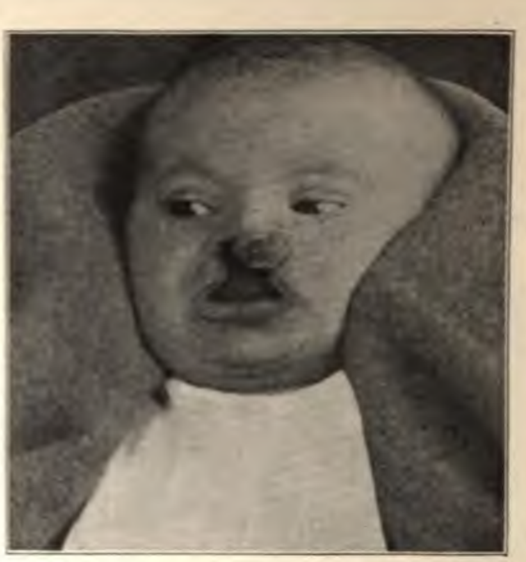


Fig. 280.—Double harelip and cleft palate. (From Eisendrath.)

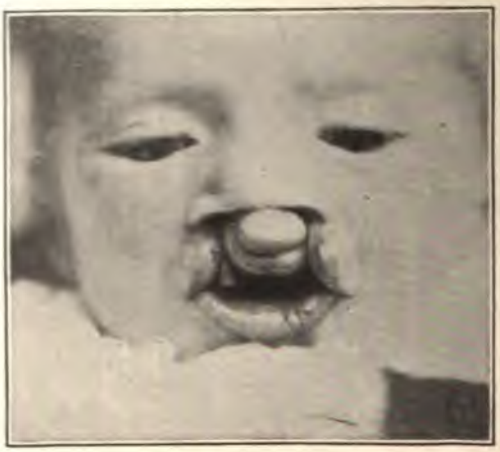
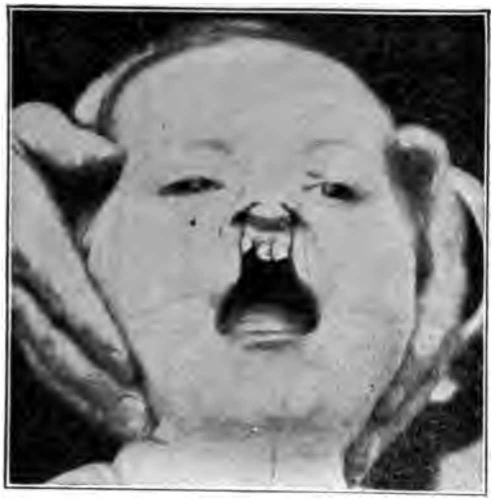
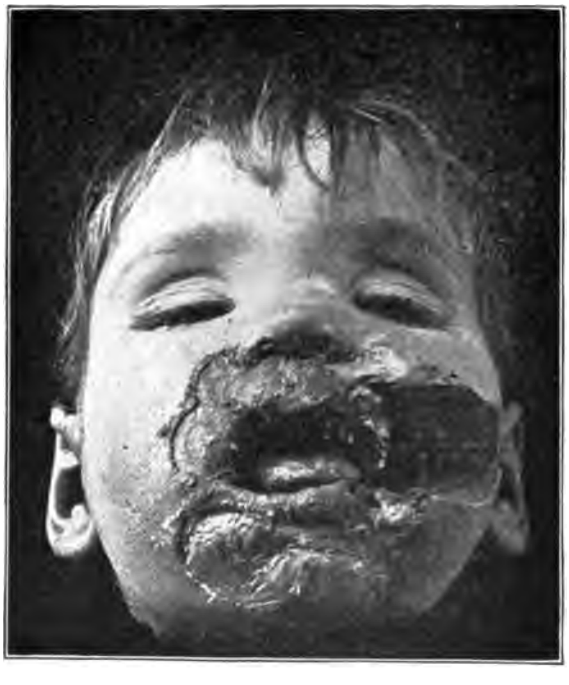


Fig. 281.—Case of complete double cleft in which at birth a tooth hung from the lateral margin of the alveolar cleft by a thin pedicle of soft tissue. (From Blair.)

Epithelioma.—A chronic irregular ulcer at the mucocutaneous junction of the lower lip in a person past middle life with enlargement of the lymph glands at the angle of the jaw, is suggestive of epithelioma.



—Complete double cleft of the lip. This is here accompanied by a
he palate. The intermaxillary bone carries three incisors. (From



3.—Noma. A piece has been removed from the left cheek for exa
(From the Hunterian Museum, London.) (From Blair.)

Hare-lip is recognized as a vertical slit or cleft in the upper lip on one or both sides of the median line. The cleft may be small and confined to the lip, or may be associated with cleft-palate, club-foot or other deformity.

Noma, or cancrum oris, is recognized as a gangrenous mass of tissue involving the lip and adjacent surface of the cheek accompanied by a very foul odor. Occurring after measles and diphtheria, it is frequently a sequence of ulcerative stomatitis.

THE BREATH

Foul Breath.—A foul breath may be caused by carious teeth, diseased gums in pyorrhea alveolaris or mercurial poisoning, follicular tonsillitis, ulcerative or gangrenous stomatitis, or gangrene of the lung.

Uremic Breath.—In uremia the breath has a urinous or ammoniacal odor.

Diabetes.—Diabetes mellitus imparts a sweetish, fruity odor to the breath, the acetone breath.

THE TEETH

Premature and **delayed** dentition possesses diagnostic significance. The former suggests hereditary syphilis, while the latter accompanies rickets, cretinism, and disorders of nutrition.

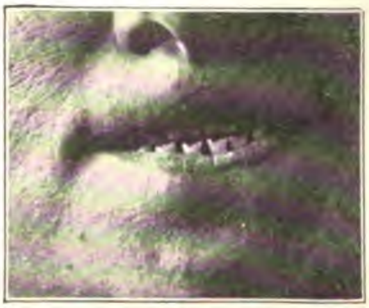


Fig. 284.—Hutchinson's teeth. (Courtesy of Drs. Fordyce and MacKee.) (From Sutton.)

Early Decay.—In children early decay of the teeth occurs in association with rickets and gastrointestinal digestive disorders. In adults carious teeth occur in pregnancy and diabetes mellitus as well as in chronic phosphorus poisoning.

Loosening of the teeth, with spongy bleeding gums, occurs in scurvy and mercurial poisoning. In pyorrhea alveolaris the teeth are loosened.

Hutchinson Teeth.—In hereditary syphilis the upper central incisors may present each a notch in its free border. These teeth are small and separated by distinct intervals. It affects the permanent and not the deciduous teeth.

Grinding of the teeth during sleep in children is observed in connection with rickets and derangements of digestion.

THE GUMS

Blue Line.—A blue line on the margin of the gum is indicative of chronic lead poisoning. In the early stages of the intoxication the line is not continuous, but occurs as a series of blue dots at the base of the teeth. The line may extend along the entire length of the gum, or may be limited to the bases of a few of the front teeth in either jaw.

In chronic copper poisoning a blue or greenish line develops along the roots of the teeth.

Red Line.—A red line along the gingivodental margin occurs with pyorrhea alveolaris, gingivitis, frequently in diabetes, and it is said in tuberculosis.

Spongy Gums.—In ulcerative stomatitis the gums are swollen, spongy, of deep red or purple color, with a line of ulceration adjacent to the incisors, sometimes extending to all the teeth.

In scorbutus the gums are spongy, bleed easily, and the teeth are loosened. In mercurial poisoning the gums are spongy, there is excessive salivation, and fetid breath.

In pellagra the gums are spongy, and assume a cerise color.

Epulis.—An epulis is a small, soft tumor springing from the gums or alveolar process of the superior maxillary bone. It is usually a giant-cell sarcoma.

THE TONGUE

Size of the Tongue.—*Hypertrophy* of the tongue occurs in infants as a congenital condition, the tongue reaching such enormous size that it cannot be contained in the mouth. *Acquired hypertrophy* of the tongue is seen in acromegaly, myxedema, and cretinism, in acute glossitis, and as a result of lymphatic obstruction, macroglossia.

Atrophy of the tongue occurs as a part of glossolabiolaryngeal palsy. Unilateral atrophy of the tongue may accompany facial hemiatrophy and as a result of hemorrhage or tumor developing in close proximity to the hypoglossal nucleus.

Movements of the Tongue.—The manner in which the tongue is protruded upon request as well as the integrity of its movements during mastication and speech should be carefully noted. Thus in nervous and neurasthenic subjects the tongue is protruded quickly upon request, whereas in typhoid states the protrusion is very slow and tardy.

In unilateral paralysis of the tongue, accompanying hemiplegia or unilateral hypoglossal palsy the tongue deviates from the median line when protruded. In bilateral paralysis of the tongue, as a result of bulbar paralysis, or symmetrical lesions of the cortex or supranuclear tracts, the tongue lies upon the floor of the mouth and cannot be protruded.

Inability to perform the finer movements of the tongue concerned in mastication and speech is an early sign of glossolabiolaryngeal paralysis, or true bulbar palsy. A similar impairment of the movements of the tongue accompanies pseudobulbar paralysis, a state in which the central lesion is not situated in the medulla, but in the lingual fibers from the cortex above the medulla. This condition of pseudobulbar palsy is not accompanied by atrophy of the tongue, which only occurs in true bulbar palsy, or glossolabiolaryngeal paralysis. The absence of lingual atrophy in the former indicates that the causative lesion is in the upper neurone, above the nucleus.

Tremor.—A coarse or fine tremor of the tongue upon protrusion accompanies many organic nervous diseases and typhoid states, and exhausting fevers. In organic nervous involvement the tremor is constant, whereas in typhoid states it only develops upon protrusion of the tongue.

Spasm.—Tonic spasm of the tongue accompanies Thompsen's disease, or myotonia congenita. A similar tonic spasm of the tongue occurs from reflex irritation of the trigeminal nerve. Clonic spasm of the tongue is noted in connection with chorea, epilepsy, puerperal melancholia, multiple sclerosis and paresis.

Ulceration.—Ulceration of the dorsum of the tongue may be indicative of simple ulceration, tuberculosis, syphilis, or carcinoma.

Simple ulceration results from local trauma or irritation. Not infrequently in young children an ulcer of the frenum is noted, resulting from the irritation of the sharp edges of the lower central incisor teeth.

A *tuberculous ulcer* of the tongue may be oval, linear, or stellate. The surface of the ulcer is pale and uneven, covered with grayish exudate, presenting no evidences of acute inflammation. It is usually accompanied by tuberculosis of the cervical lymphatic glands.

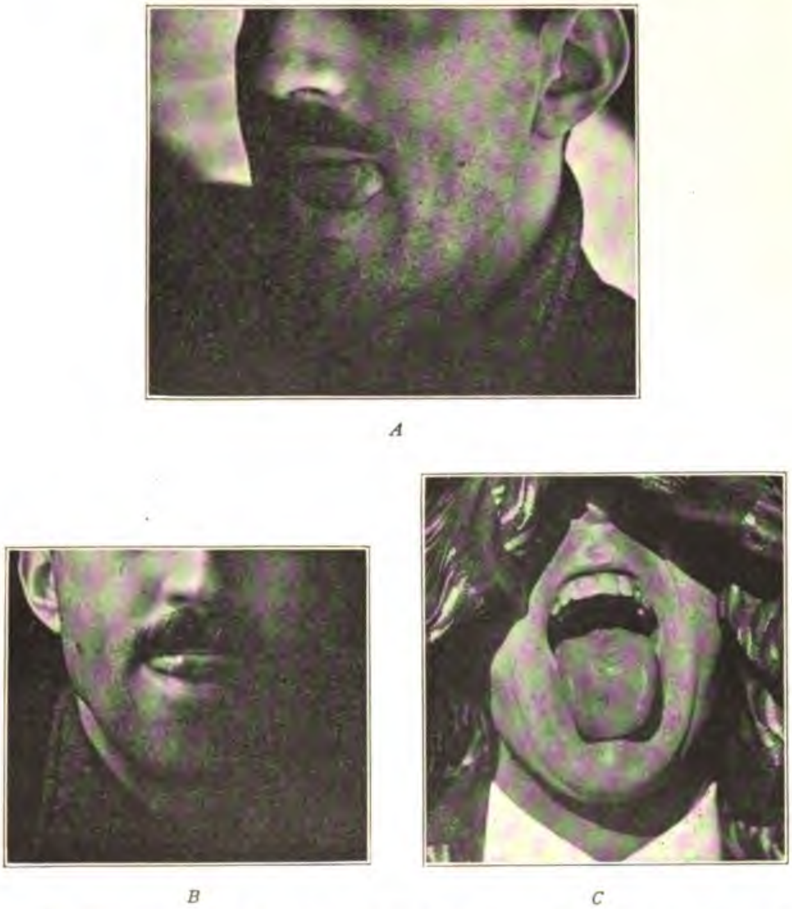


Fig. 285.—Illustrating tuberculous lesions of the tongue, *A* and *B* in the same individual, healing after two and one-half years' treatment; *C*, on the dorsum of the tongue of an individual suffering from active advanced tuberculosis; no signs of healing shown, patient dying of the disease a few months later. (From Pottenger.)

A *syphilitic ulcer* of the tongue produces a chronic dissecting glossitis characterized by multiple fissures situated principally upon the lingual edges, but crossing the dorsum of the tongue in various directions. The cervical lymph glands are commonly enlarged.

A *carcinomatous ulcer* of the tongue is solitary, with induration of the surrounding tissues, somewhat simulating a chancre; but the ulcer does not disappear under antisyphilitic therapy. The carcinomatous ulcer is apt to develop in elderly persons, and the accompanying glandular enlargement appears more tardily than in the other lingual ulcers.

Geographical Tongue.—In the geographical tongue there are one or more patches upon the dorsum in which the surface epithelium has desquamated, the patches extending at the periphery while healing at the center, pursuing a circinate course over the dorsum, two or more patches frequently coalescing. Occurring in undernourished children and adults, the geographical tongue possesses little significance.

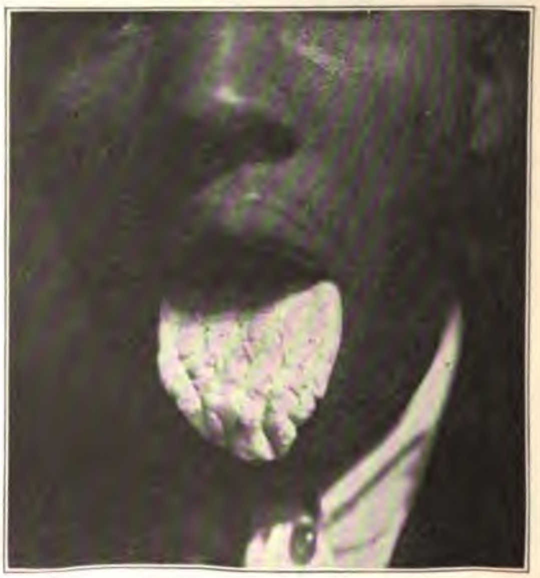


Fig. 286.—"Cobblestone tongue" due to gummous deposits two years after infection. (From Hazen.)

Leucoplakia.—In leucoplakia irregularly shaped plaques of thickened epithelium appear upon the dorsum of the tongue. The plaques are smooth, pale, slightly elevated above the surrounding surface of the tongue, one to two centimeters in diameter, and nonulcerative. Their recognition is important in that they have become the starting point of carcinoma of the tongue.

Smoker's Patch.—In persons who use tobacco to excess a roun
or oval patch is sometimes

tongue near the tip, slightly elevated, red or pearly in color, smooth, and with no tendency to ulceration.

Cysts.—Mucous and blood cysts occasionally develop in the tongue. Rarely the cysticercus cellulosæ, the larva of the tænia solium may produce a cyst upon the under surface of the tongue, or an echinococcus cyst may be encountered in this region. Also a ranula, due to obstruction and dilatation of the ducts of Blandin-Nuhn's glands may be found on the under surface of the tongue near the tip.

Thrush.—In thrush the dorsum of the tongue is covered or studded with small, white flakes, resembling closely deposited flakes of coagulated milk, but differing from them in that they cannot be wiped off; and, if removed, leave bleeding points.

Indentation of the edges of the tongue by the teeth, is noted during prolonged fevers when the hygiene of the oral cavity is not properly practiced. In pellagra there are similar indentations in the deep red border of the tongue occurring with this disease.

Pellagra.—In pellagra the tongue presents a fiery red border and tip, showing indentations corresponding to the teeth with which it is in contact. Frequently small circumscribed sloughs are encountered upon the borders of the tongue, corresponding to areas of epithelial denudation. The gums are spongy and of a cerise color, while the mouth is the site of a stomatitis of variable intensity. The condition of the tongue and buccal mucosa is intensely painful.

Dryness of the Tongue.—The tongue in health is kept moïstened with the salivary secretions and the buccal secretions. When this secretion is inhibited during acute fevers, the administration of atropine, or from excessive loss of body fluids incident to prolonged diarrhea and profuse hemorrhage the tongue is abnormally dry, and not infrequently covered with a thick brown coat.

Color.—The ingestion of various chemicals and drugs and certain diseases alter the color of the tongue. The tongue is white following the ingestion of mercuric chloride, ammonia, sulphuric acid and phenol. The ingestion of caustic potash or soda causes reddening of the tongue with evidences of destructive action of these substances. Hydrochloric and nitric acids color the tongue yellow.

In Addison's disease and purpura hemorrhagica the tongue not infrequently exhibits small nonelevated purple spots; while xanthelasma produces yellowish, slightly elevated dots along the margins of the tongue.

In scarlatina the tongue is bright red, the filiform papillæ contrasting sharply with the slight white furring of the tongue, the *strawberry tongue.*

THE BUCCAL CAVITY

Color.—The buccal mucosa is pallid in anemic states, is excessively red during local inflammation as in catarrhal stomatitis, and is bluish in cyanosis and argyria.

Moisture.—Excessive moisture of the buccal cavity, incident to overactivity of the salivary and buccal glands accompanies local inflammation, following the ingestion of massive doses of the iodides and mercurial salts, during the early stages of smallpox and typhus fever, occasionally during pregnancy, and during convalescence from typhoid fever.

Dryness of the buccal cavity, or xerostomia, owing to temporary arrest of the salivary and buccal secretions occurs during acute febrile diseases, diabetes mellitus, in mouth breathers and lesions of the pons and medulla affecting the integrity of the nervous mechanism of the salivary glands.

Eruptions.—In variola and varicella vesicles may appear upon the buccal mucosa, similar vesicular eruptions accompanying herpes buccalis and aphthous stomatitis. Measles is accompanied by pathognomonic lesions upon the mucosa, *Koplik's spots.* These are minute red spots with a bluish-white center, occurring upon the inner surface of the cheek opposite the molar teeth. The number of the spots varies; there may be only one or two or the mucosa may be fairly studded with them. The spots occur early, disappearing with the inception of the exanthem.

Mucous Patch.—The mucous patch of syphilis is frequently encountered upon the buccal mucosa. In all suspicious cases a careful search of the mucous lining of the cheek should be made for these lesions.

Noma, cancrum oris, or gangrenous stomatitis, develops as an indurated spot upon the mucous lining of the cheek near the angle of the mouth, later involving the entire thickness of the buccal wall, the gangrenous tissue emitting an especially foul odor.

THE PHARYNX

In examining the pharynx the tongue should be gently depressed with a wooden spatula or other type of tongue depressor, while the patient is instructed to utter the word "AH," which lowers the base of the tongue, permitting a good view of the posterior pharyngeal wall.

Redness.—Abnormal redness of the pharyngeal wall accompanies acute inflammation of the pharynx, which may be primary, or occur with the acute exanthematous fevers, or acute infectious disease, as influenza and erysipelas.

Eruptions.—The eruptions of variola, varicella, and of herpes buccalis are often distributed generally over the pharyngeal wall.

Ulceration of the pharyngeal wall is indicative of tuberculosis, syphilis, or typhoid fever.

Bulging of the posterior pharyngeal wall, either in the median line or laterally, occurs in postpharyngeal abscess, which is often due to tuberculous disease of the cervical vertebræ.

Elongated Uvula.—Elongation of the uvula may occur from inflammation of the adjacent pharyngeal mucous membrane, but it may also be a part of the general edema incident to cardiac or renal disease.

Perforation of the soft palate is usually of syphilitic origin.

Paralysis of the soft palate may be unilateral or bilateral. Paralysis of the palate is detected by observation of its movements while the patient speaks, at which time the normal palate moves upward. If this normal mobility of the palate is lost on one side, the paralysis is unilateral; if both sides remain immobile, the paralysis is bilateral. Bilateral palatal paralysis is not infrequently attended by regurgitation of fluids through the nose upon the attempt to swallow. Paralysis of the palate may be part and parcel of glossolabiolaryngeal paralysis, may depend upon cervical caries, or may be due to diphtheritic paralysis.

THE TONSILS

Inflammation.—A moderate grade of tonsillar inflammation resulting in painful deglutition, accompanies most of the acute exanthematous fevers. In acute follicular tonsillitis the tonsils are moderately enlarged, red, and studded with minute yellowish dots, corresponding to plugs of mucus, epithelium and bacteria which can be squeezed from the tonsillar crypts.

Chronic simple enlargement of the tonsils, in which the two bodies may almost meet in the median line, occurs occasionally in childhood.

Pseudomembrane.—A pseudomembrane upon the tonsil, perhaps involving the pharyngeal wall as well, if not diphtheria, is apt to be due to streptococcic inflammation or scarlatina.

Ulceration.—Ulceration of the tonsil is due to tuberculosis, syphilis, or, if in an elderly person, to carcinoma, or in a younger subject to sarcoma.

Peritonsillar Ulceration (Vincent's Angina)—In this disease which is a unilateral affection, there is ulceration of the peritonsillar tissues, with a variable amount of yellowish exudate covering the tonsil. There is marked swelling of the submaxillary lymph glands.

CHAPTER XXIX

EXAMINATION OF THE NECK

Shape.—Certain variations in the shape of the neck characterize certain diseases. Thus, a short, thick neck suggests hypertrophic emphysema, and is a constant accompaniment of the barrel chest of this disease. Similarly in plethoric patients the neck is short and thick. A long, slender neck, on the other hand, is frequently observed in phthisical patients.

Rigidity.—This may be caused by tuberculous disease of the cervical vertebræ or rheumatism of the muscles of the neck. Tender, enlarged cervical glands or boils or carbuncles may cause the patient to hold the neck rigid. As previously stated, retraction and rigidity of the neck occur in meningitis, tetanus, and strychnine poisoning. Rigidity of the neck with fixation of the head is also observed as a result of arthritis deformans, spasmodic torticollis, and due to scars from extensive burns of the neck.

Prominent Sternomastoids.—Abnormal prominence of both sternomastoid muscles is usually a sign of long continued dyspnea, due to pulmonary or cardiac disease. An undue prominence of one sternomastoid may be caused by spasmodic torticollis, a tumor, cyst, or abscess of the muscle.

Torticollis.—This is a spasm, usually tonic, rarely clonic, of the sternomastoid and trapezius muscles. Its cause is occasionally irritation of the spinal accessory nerve which supplies these muscles, by cicatrices or enlarged glands. Most cases, however, occur without assignable cause. Congenital torticollis is caused by congenital shortness of one sternomastoid and is not due to spasm in any sense.

Deflection of Larynx and Trachea.—The larynx and trachea, the latter overlaid by the thyroid gland, occupy the median line of the neck. Deflection of these structures to one or the other side may be due to atrophy of the muscles on one side of the neck, to tumor or aneurysm in the adjacent tissues, or to disease of the thoracic viscera. Of the last named factors, fibroid phthisis is very important, the structures being deflected toward the side of the cirrhotic lung.

Movements of the Larynx and Trachea.—Marked inspiratory descent of the larynx occurs in laryngeal stenosis. Normally the larynx descends slightly during inspiration and rises to a similar degree during expiration. When this mobility is abolished in a dyspneic patient the cause of the dyspnea is below the larynx as, for instance, pressure on a bronchus by enlarged glands or aneurysm.

Tracheal Tug.—This is an important sign of aneurysm of the thoracic aorta, and has been discussed in a previous section.

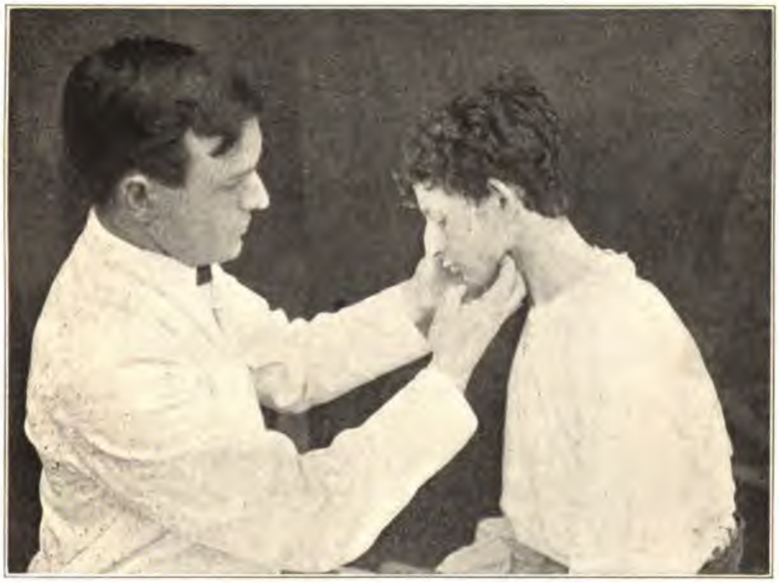


Fig. 289.—Palpation of submaxillary and submental glands. (From Eisendrath.)

Thyroid Gland.—This gland may be increased in size or it may be diminished in size.

Enlargement of the thyroid may involve one or both lobes. The degree of enlargement varies. There may be a small localized swelling at one point, or the entire gland may be found greatly enlarged, exerting dangerous pressure upon the trachea, carotid arteries, and nerves. The consistence of the enlarged gland varies. In the fibrous forms of goiter the gland is hard, while in the cystic form it is soft and may fluctuate. Sometimes a thrill may be detected on palpating the gland, accompanied by a systolic murmur, due to the increased vascularity of the gland. An enlarged thyroid gland moves with the trachea during deglutition.

The significance of a thyroid enlargement varies with the cause. It may be due to abscess following an infectious disease, or to malignant growth. If due to simple hypertrophy of the gland, the tumor will usually appear during pregnancy, and disappear spontaneously after labor. If fluctuation is detected, it is probably a cystic goiter or an abscess of the gland. If the enlargement be due to exopththalmic goiter it will be associated with the cardinal symptoms of this disease, as tachycardia, exophthalmos, and tremor.

Atrophy of the thyroid gland, revealed by the presence of a depression in the normal position of the gland, occurs in cretinism and myxedema.

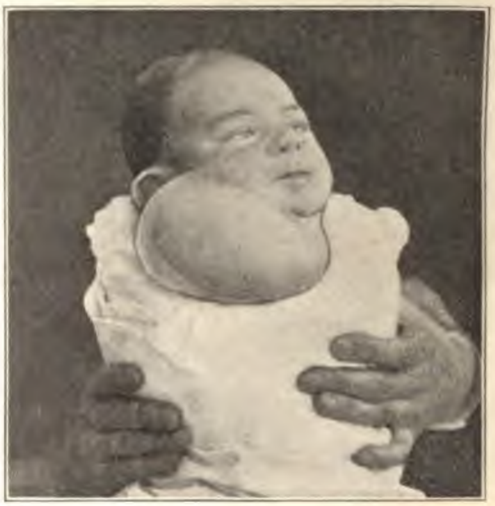


Fig. 290.—Congenital hemangioma of neck. (From Eisendrath.)

Enlarged Glands.—Enlarged lymph glands in the cervical region may have a varied significance, the significance varying with the location of the glands involved and with the state in which they are found; namely, whether hard or soft and fluctuating, whether single and individual or matted together in a mass. Among the causes of glandular enlargement in this region may be mentioned the following conditions.

The lymph glands at the angle of the jaw may enlarge from follicular tonsillitis, diphtheria, scarlatina, measles, German measles, varicella and smallpox; also in erysipelas, glanders, whooping cough, and retropharyngeal abscess. In these conditions the glands are acutely tender for a period and usually undergo resolution without abscess formation.

The submental glands, just below the chin may enlarge as a result of carious teeth, stomatitis, syphilis, mumps, cancer of the lower lip or anterior portion of the tongue. This group of glands is often enlarged in cases of actinomycosis.

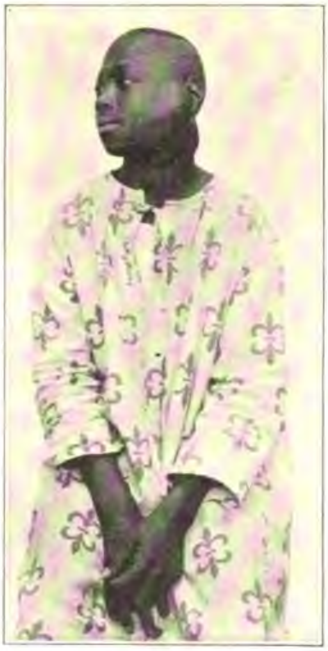


Fig. 291.—Hodgkin's disease.

The parotid lymph glands enlarge in mumps, and diseases of the upper pharynx and skin of the face, as well as in malignant disease of the parotid gland.

The occipital glands are enlarged in German measles, often the only group attacked in this disease. Enlargement of this group of glands is also a valuable sign of syphilis, and occurs also in cases of pediculosis of the scalp.

Tuberculosis of the cervical glands causes glandular enlargement, particularly in the glands under the jaw. The glands tend to become adherent to the cutaneous structures and often suppurate.

Hodgkin's disease causes glandular enlargement in the lymph glands of the neck, of long standing, involving also the glands of the axilla, groin, and showing slight splenic enlargement.

Lymphatic leukemia is a cause of enlargement of the cervical lymph glands.

Enlargement of the lymph glands above the left clavicle points to cancer of the stomach.

The condition of the glands and the duration of the enlargement possess diagnostic significance. Thus, acute painful cases of short duration are probably due to a tonsillitis, or the exanthemata. Chronic cases, of long standing, may be due to tuberculosis, syphilis, or Hodgkin's disease. In tuberculosis the glands are matted together with tendency to suppurate. In syphilis they are hard and small. In Hodgkin's disease the glands are enlarged, but remain separate, and do not tend to suppurate.

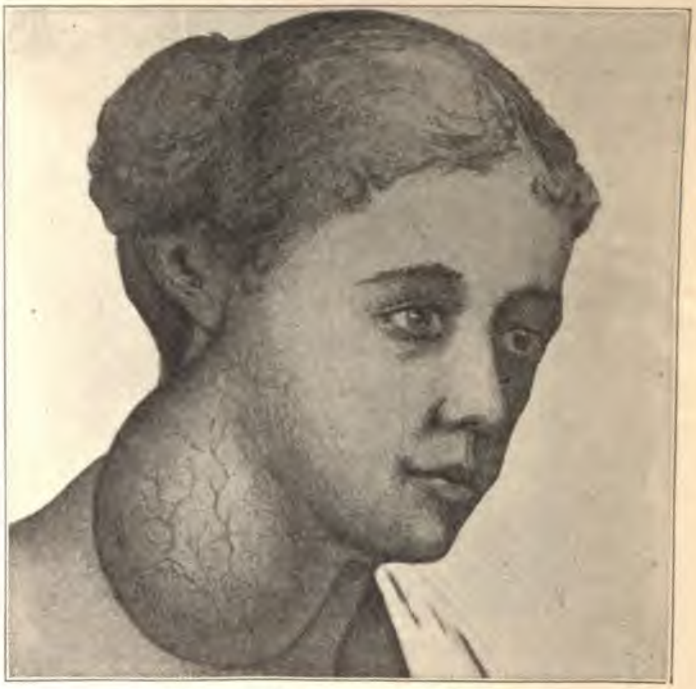


Fig. 292.—Branchial cyst. (From McFarland.)

Abscess.—An abscess in the cervical region is almost certainly of tuberculous origin, being the result of tuberculosis of the cervical lymphatic glands or of Pott's disease.

Scars.—Scars in the neck are usually the result of cervical tuberculosis, surgical procedures or trauma.

Branchial Cysts and Fistulæ.—Branchial cysts and fistulæ, resulting from imperfect closure of the embryonic branchial clefts, are encountered in rare instances. A branchial cyst is formed by closure of the pharyngeal and cutaneous surfaces of the cleft without closure of the intervening mesoblastic tissues. Branchial fistulæ may be complete or incomplete, depending upon the degree of fusion of the embryonic clefts, the incomplete being represented by diverticula, either external or internal, opening into the pharynx.

Ludwig's angina, a painful indurated swelling beneath the angle of the jaw, due to septic infection of the tissues surrounding the submaxillary gland, is an occasional cause of dyspnea and dysphagia which may become alarming.

Woody or ligneous phlegmon, an insidious induration of the subcutaneous tissues of the neck, involving the lateral or anterior aspect of the cervical region, is occasionally encountered. In some instances the structures are indurated from the jaw to the clavicle; the condition is attended by little pain and no fever.

SECTION II

EXAMINATION OF THE HAND AND ARM

CHAPTER XXX

THE HAND

THE NAILS

Pallor.—Pallor of the nails is a sign of anemia, and it is well to bear in mind the rule laid down by Stephen McKenzie, that when pressure upon the tip of the finger completely drives the blood from beneath the nail, the red corpuscles are present in only half their normal number.

Cyanosis.—Cyanosis or blueness of the nails is a sign of deficient aeration of the blood, either due to a failing heart, an obstructive pulmonary lesion, or the ingestion of coal tar products. As has been stated, cyanosis appears very early under the nails and about the lips.

White spots in the nails are usually significant of trophic changes in the nail; less commonly they are due to injury of the matrix by picking at the base of the nail.

Capillary Pulse.—The capillary pulse has been described under the section upon the examination of the circulatory organs; and as stated, is a valuable sign of aortic regurgitation or Corrigan's disease.

Transverse Groove.—A transverse groove on the back of a nail is a sign of a recent acute illness. The groove has its inception at the base of the nail and its distance from the base when observed may indicate when convalescence from the illness in question commenced. Thus it requires six months for the groove to progress from the base to the free edge of the nail; hence, if it be encountered half way between the matrix and the free edge, it is an indication that convalescence began approximately three months previously.

Longitudinal ridges in the nails are said to be a reliable sign of gouty diathesis. Certainly the ridges are encountered in many gouty patients.

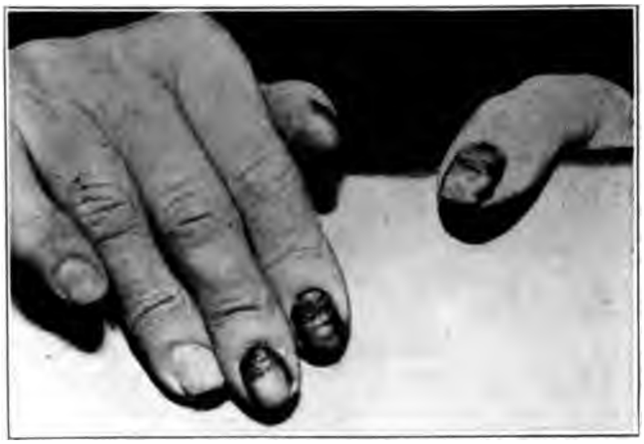


Fig. 293.—Hypertrophy of the nails. (From Hazen.)

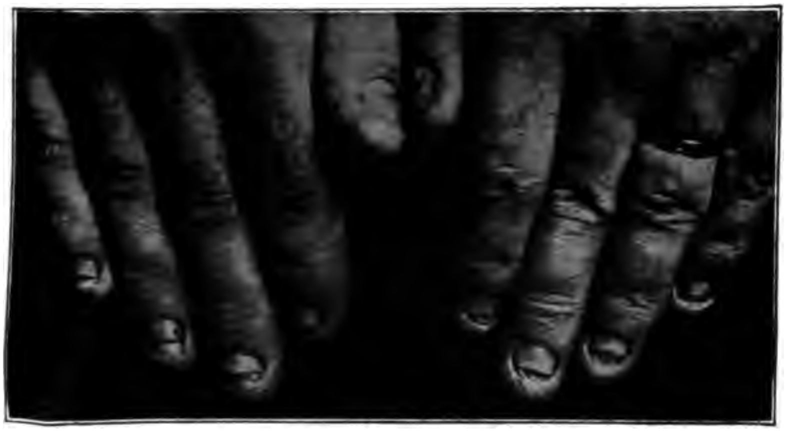


Fig. 294.—Symmetrical atrophy of nails. (Courtesy of Dr. J. C. E. King and Dr. H. G. Parker.) (From Sutton.)

Incurvation of the nails, with or without clubbing of the finger-tips is a sign of chronic disease of the heart or pulmonary tissues, such as cardiac failure, aneurysm, phthisis, or emphysema.

The incurvation may be lateral or longitudinal, or may occ
both directions.

Hypertrophy of the nails, particularly in the transverse
rection associated with thickening and sometimes with twisti
occurs after acute fevers, particularly following typhoid fev
in connection with syphilis, and in sclerodactyly. A similar h
pertrophy of the nail may result from eczema, may be encou
tered in a subject with Raynaud's disease, and in pulmonar
osteoarthropathy. The nail may be simply hypertrophied with
out any defect in its structure (megalonychosis); or in addition
to hypertrophy the nail may be twisted spirally (onychogry-
posis).

Atrophy of the nails, with ulceration at the base, occurs in Mor-
van's disease, a syndrome which develops as a sequence of neuritis
and syringomyelia. Atrophy of the nail may follow psoriasis of
the fingers.

Arrested Growth.—The growth of the nails is impaired or
ceases on the paralyzed side in hemiplegia. A similar arrest of
growth of the nails of the paralyzed limb occurs in infantile paral-
ysis. Arrest of growth of a nail may be detected by staining the
nails at identical points upon the two hands and observing any
discrepancy between the growth of the nails.

Excessive brittleness of the nails is noted in persons of gouty
liathesis, the nails frequently presenting the longitudinal stria-
ions which have been described.

Onychia, ulceration of the nail matrix, occurs in children with
reditary syphilis, or scrofula, and it is said in persons who are
dicted to the chloral habit.

aronychia, or whitlow, an acute inflammation of the tissues
rounding the matrix of the nail, may be a sequence of local
ma or may be caused by lateral hypertrophy of the nail.

dolent Sore.—An indolent sore near the root of the nail, if
rated and associated with enlargement of the epitrochlear
h glands, is usually a chancre; but may be due to tubercu-

THE FINGERS

.—Tophi are concretions of sodium biurate which occur in
ts of the fingers in gouty subjects. They are more promi-
the dorsal surface of the joints, and may break throu

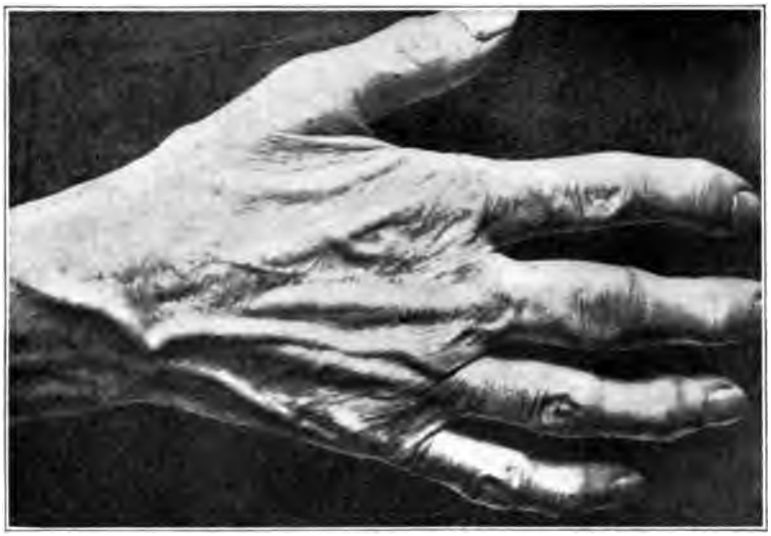
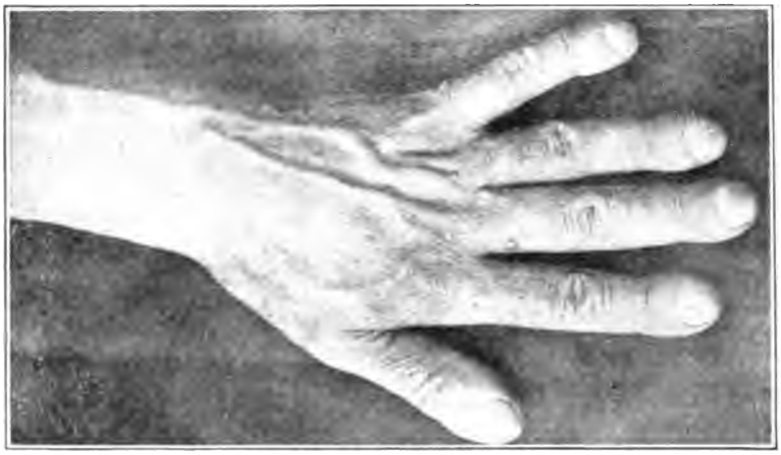


Fig. 295. Heberden's nodes. (From Butler.)



the skin, when they constitute the "chalk stones" of the disease.

Enlarged Joints.—Enlargement of the joints of the fingers is seen in connection with gout and chronic rheumatism. In rheumatism the enlarged joints are often hot and painful.

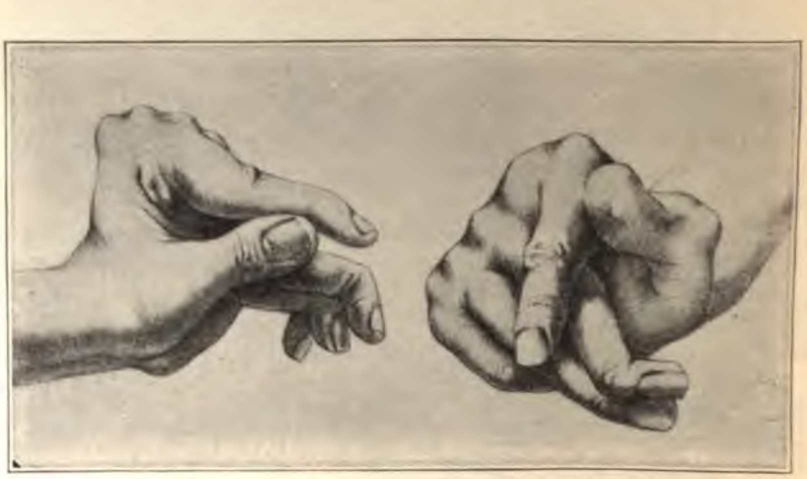


Fig. 297.—Arthritis deformans. (From Butler.)

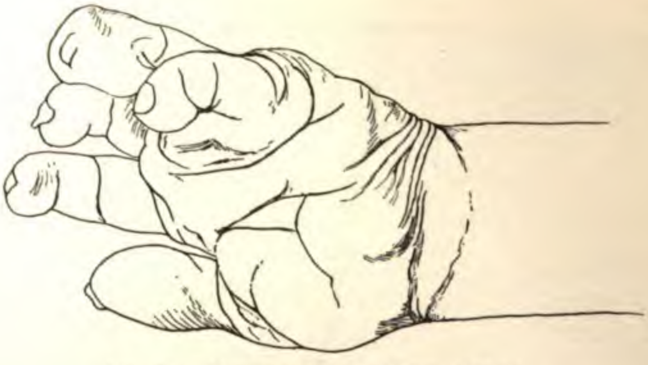


Fig. 298.—Morvan's disease. (From Butler.)

Heberden's Nodes.—These nodes, also termed Haygarth's Nodosities, are knobby enlargements of the proximal ends of the terminal phalanges. They are noted in gout and in arthritis deformans, in which diseases they are said to be of good prognostic significance.

Clubbed Fingers (Hippocratic Fingers).—Clubbing of the terminal phalanges accompanies many chronic diseases of the heart and lungs, notably chronic bronchitis, emphysema, phthisis, and chronic pleurisy, and uncompensated cardiac disease. The nails are commonly incurved. An exaggeration of this condition with swelling of the carpal joints is noted in pulmonary osteoarthropathy.

Distortions of the fingers accompany gout, chronic rheumatism, and arthritis deformans. The distortions are not produced merely by fixation of the fingers in abnormal positions, but are produced by organic changes, in gout by the deposition of sodium biurate in the joints, in arthritis deformans by absorption of bone and the growth of exostoses. The fingers are most frequently deflected toward the ulnar side of the hand.

Dactylitis.—Dactylitis is usually a sign of hereditary syphilis, less frequently of tuberculosis. In the evolution of the deformity a fusiform purple swelling, which is prone to undergo ulceration with sinus formation, appears upon one or more of the fingers, most frequently involving the proximal phalanges.

Raynaud's Disease.—In Raynaud's disease, or "dead fingers" the fingers are bluish-black or livid, gangrene occurring in spots and leading in many instances to spontaneous amputation of the fingers.

Morvan's Disease.—In this disease the fingers are the site of painless, destructive whitlows, which have their inception adjacent to the base of the nail, leading to necrosis of the terminal phalanges and marked swelling of the fingers.

SHAPE OF THE HAND

The shape of the hand varies in different subjects and under varying conditions of age and occupation. The broad, heavy hand is said to be indicative of a sanguine personality, while the slender hand is said to indicate a nervous temperament. Bluish dotting of the hand of a coal miner points to the possibility of anthracosis, while in old age and in malignant disease and phthisis the hand is small and withered.

Spade Hand.—In acromegaly and myxedema the hand is large, with thick fingers and broad nails. In myxedema the hand is boggy, but does not pit on pressure; whereas in acromegaly the hand is hard, as the basis of the hypertrophy is osseous.

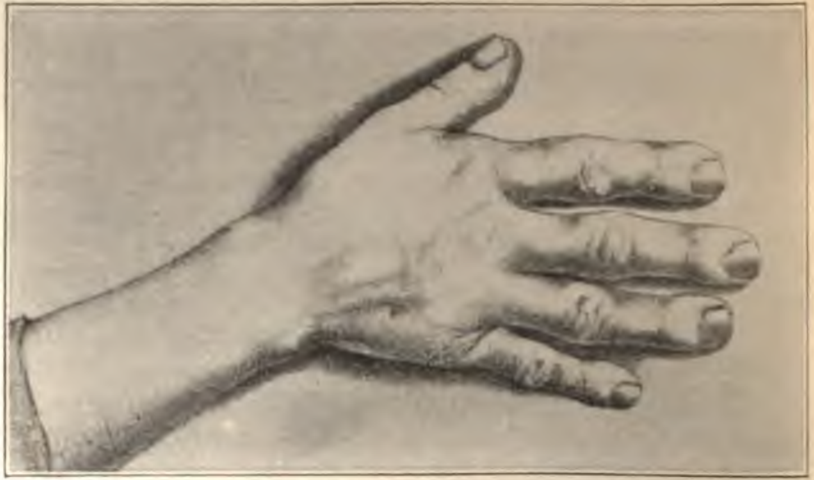


Fig. 299.—Spade hand. (From Butler.)

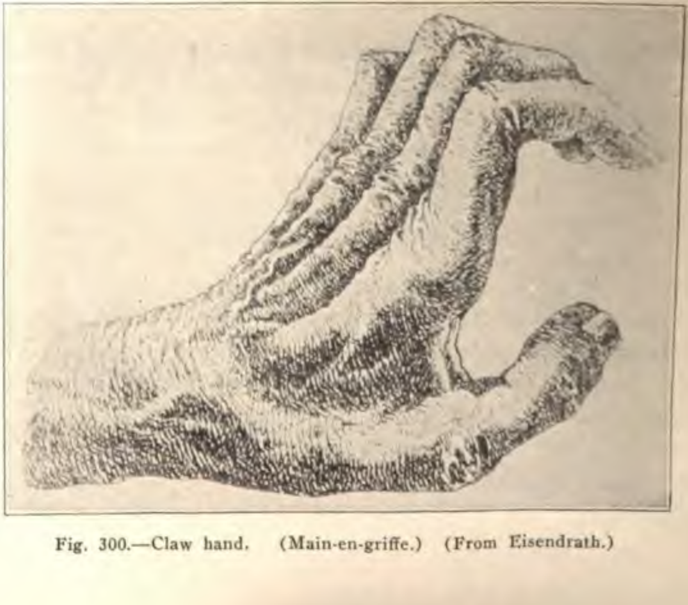


Fig. 300.—Claw hand. (Main-en-griffe.) (From Eisendrath.)

Claw-Hand (Main-en-griffe).—In amyotrophic lateral sclerosis and progressive muscular atrophy the proximal phalanges are drawn backward toward the wrist, while the second and third phalanges are flexed toward the palm. The underlying cause of this deformity is a paralysis of the lumbrical and interosseous muscles, causing the proximal phalanges to assume a state of dorsal extension, while the distal phalanges are flexed.

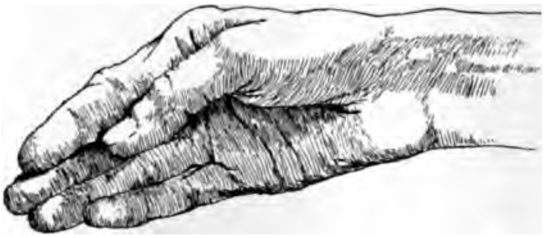


Fig. 301.—Accoucheur's hand. (From Butler.)

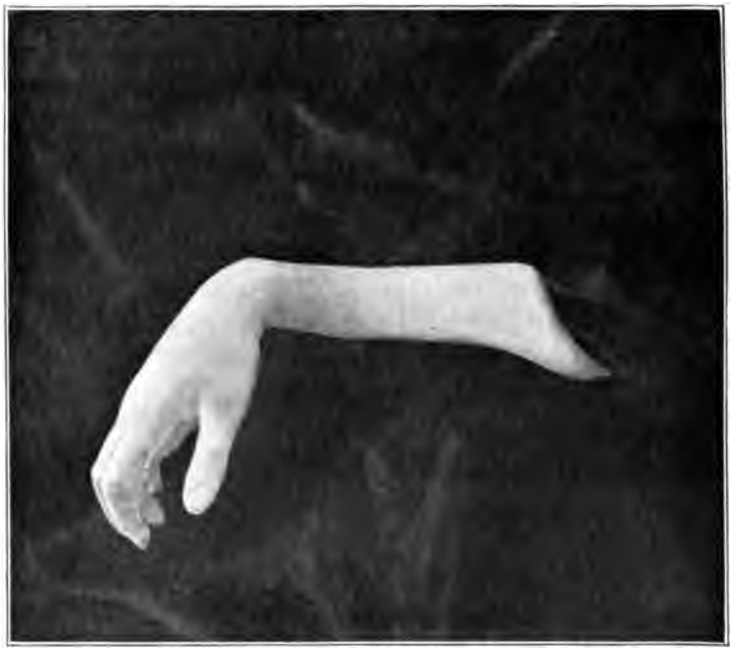


Fig. 302.—Wrist-drop. (From Eisendrath.)

Hemiplegic Hand.—In hemiplegia the contractures of the arm and hand are replaced in course of time by permanent deformity. The fingers in this deformity are flexed upon the palm, the wrist is flexed upon the forearm, while the elbow is retained in a state of permanent flexion and applied closely to the side of the body.

Seal-fin Hand.—In chronic gout and rheumatoid arthritis the entire hand is deflected toward the ulna as a result of spasm of the extensor muscles, imparting to the hand a fancied resemblance to the fin of a seal.

Ape Hand.—This type of manual deformity is the result of wasting of the thenar and hypothenar muscles in progressive muscular atrophy, causing the hand to assume a position in which the fingers and thumb are on one parallel plane.

Accoucheur's Hand.—In this type of manual deformity, which occurs in tetany, the thumb is flexed into the palm of the hand, while the fingers, flexed at the metacarpophalangeal joints and first interphalangeal joints, are extended at the second interphalangeal articulations and pressed closely upon the thumb.

Dupuytren's Contracture.—This is a permanent painless flexure of one finger of one or both hands into the palm. Most commonly the little finger alone is involved, but sometimes the ring finger or other fingers are flexed also. Dupuytren's contracture results from burns or other injuries to the palmar fascia.

Ganglion.—A ganglion is recognized as a localized swelling upon the dorsum of the hand. It is presumably caused by cystic degeneration of a synovial fringe within a tendon sheath. Ganglia are not infrequently tuberculous in origin.

Wrist-drop.—In wrist-drop the hand hangs powerless from the wrist. It is significant of neuritis or paralysis of the musculospiral nerve.

TREMOR OF THE HAND

Intention Tremor.—Intention tremor is a tremor of the hand which is converted into coarse shaking movements when the patient endeavors to perform any act, such as bringing a glass of water to the lips, or holding a pen to write. Intention tremor is a cardinal sign of multiple sclerosis and is sometimes noted in hysteria.

Paralysis Agitans.—In paralysis agitans the patient is subject to a constant [illegible] which the thumb and index finger are [illegible] in actual contact, describing a rolling movement as if they were rolling a pill (Pill-rolling tremor). The tremor in marked contrast to that of multiple sclerosis disappears completely during voluntary movements of the hands.

Professional Spasm.—Writers, violin players, and others who constantly employ one set of muscles are often troubled with painful spasms in the muscles used, incapacitating them for their usual occupations.

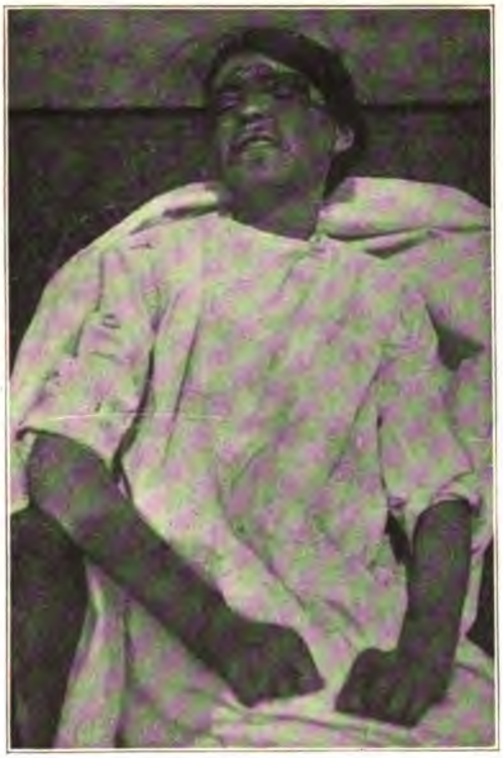


Fig. 303.—Pellagra.

Athetosis.—This term refers to certain slow and purposeless movements of the fingers which are encountered in patients suffering with organic disease of the central nervous system.

Pellagra.—The cutaneous manifestations of pellagra involve the extensor aspect of the hand and forearm, producing an eruption of an erythematosquamous type. In incipient cases, in the stage of initial erythema, the eruption resembles closely ordinary sunburn or solar erythema. In the latter evolution of the disease the superficial epithelium takes on a brown pigmentation, and desquamates in scales. Sensation is lost in the areas of desquamation. The skin covering the elbows should always be inspected for the eruption. The eruption may involve the dorsum of the foot or the face, and rarely the neck or chest.

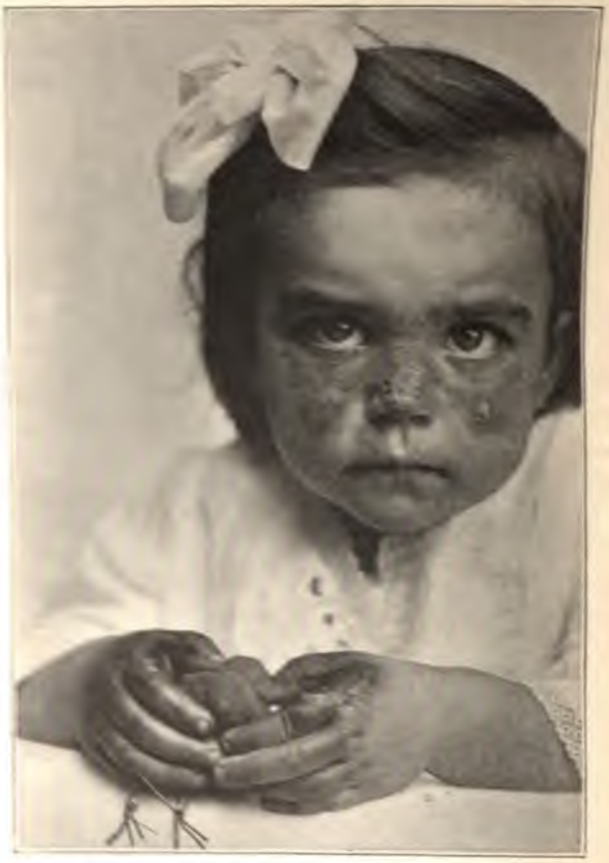


Fig. 304.—Pellagra in child less than 3 years old.

CHAPTER XXXI

THE FOREARM AND ARM

EXAMINATION OF THE FOREARM

Epiphyseal enlargement of the forearm bones at the wrist is indicative of rickets. It is usually accompanied by the rachitic rosary and other signs of the disease.

Enlargement of the lower end of the radius with clubbing of the fingers occurs in pulmonary osteoarthropathy the result of chronic pulmonary or cardiac disease.

Enlargements or nodes along the shaft of the radius or ulna are usually due to syphilitic periostitis.

Erythema nodosum occasionally occurs upon the forearm, manifesting itself by the appearance of elevated, red, shining nodular swellings, which are very painful upon pressure.

Edema of the forearm, usually affecting the arm as well, results from thrombosis of the axillary vein, or from the pressure of mediastinal tumors upon the subclavian vein.

EXAMINATION OF THE ARM

Tumors.—A superficial tumor arising in the arm is most apt to be lipomatous. It is often lobulated. A ruptured biceps produces a sharp tumor over the lower portion of the arm. A deeply seated tumor of the arm is most likely to prove sarcoma of the humerus. An acute painful swelling of the humerus following typhoid fever or scarlatina is due to acute periostitis.

Small nodular elevations upon the humerus are usually the result of syphilitic periostitis.

Paralysis.—Paralysis of one arm may be total, the arm hanging limply, without power of movement, or may be partial. Partial brachial paralysis may assume one of two types; namely, the upper arm type of Duchenne-Erb, or the lower arm type of Klumpke. Brachial paralysis may be the result of trauma at birth, or may result from compression of the brachial plexus by a tumor or by a crutch. Some cases are due to a faulty position of the arm during anesthesia. The upper arm type of Duchenne-Erb involves the deltoid, brachialis anticus, triceps, supinator longus, supinator brevis, and the infraspinatus muscles. The patient is unable to adduct the arm, and the forearm remains in a position of extension and pronation. The lower arm type of Klumpke involves the small muscles of the forearm and hand, with inability to move the hand or fingers.

Rigidity and Contracture.—In hemiplegia the paralysis of the arm is spastic and is followed in the course of time by permanent contracture. The elbow is maintained in a state of semiflexion, the wrist is flexed upon the forearm, while the arm is often closely apposed to the trunk. Spastic rigidity of the arm is often one of the earliest signs of chronic hydrocephalus.

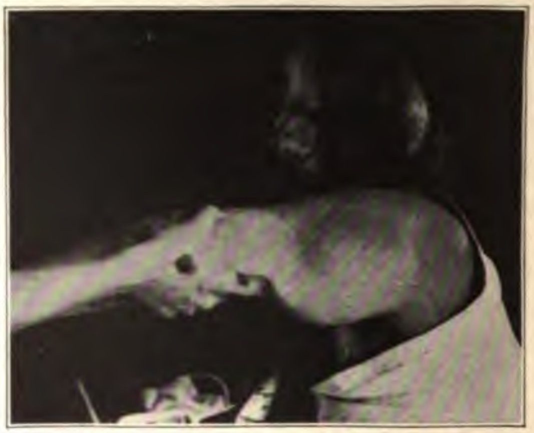


Fig. 305.—Lipoma of arm.

Movements.—In Sydenham's chorea the arms participate in the purposeless movements of the head and face. In this form of chorea there is no motor weakness; whereas in the so-called *hemiparalytic chorea,* which is attended by similar purposeless movements of the arms, the muscular power is usually impaired. Pregnant women occasionally exhibit similar purposeless movements of the arms in the so-called *chorea gravidarum.*

In *paramyoclonus multiplex* there is frequently noted a symmetrical, bilateral, clonic spasm of the muscles of the arms. The biceps, triceps, and deltoid muscles are involved. The paroxysm is characterized by a series of very rapid clonic contractions of symmetric groups of muscles in the two arms, the contractions often exceeding a hundred in a minute. Usually of very brief duration, the paroxysm may in some cases last for several moments.

Atrophy.—Atrophy of the muscles of the arm follows the paralysis of acute anterior poliomyelitis and brachial palsies, conditions which involve the lower motor neurone.

Miner's Elbow.—In this condition there is a swelling overlying the olecranon bursa, produced by chronic bursitis of this structure, which sometimes yields fluctuation on palpation.

SECTION III

EXAMINATION OF THE LOWER EXTREMITIES

CHAPTER XXXII

THE FOOT, LEG, AND THIGH

THE TOES

Gangrene of the toes is usually significant of diabetes, arteriosclerosis, or Raynaud's disease. Gangrene of the toes is less frequently a sequence of frostbite, local trauma, ergotism, or embolism in connection with cardiac disease.

Perforating Ulcer.—The perforating ulcer, or Mal Perforante, occurring with locomotor ataxia and rarely with diabetes, is a deep circular ulcer, usually situated upon the under surface of the great toe.

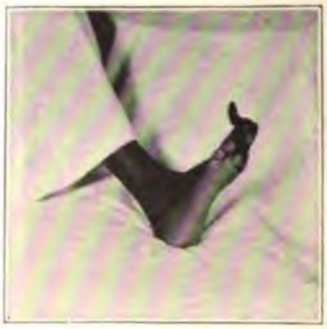


Fig. 306.—Gangrene of toes.

Gout produces hot tense swelling of the metatarsophalangeal articulation of the great toe, which is very sensitive to pressure.

THE FOOT

Flat-foot, pes planus, is a flattening or giving way of the normal arch of the foot as a result of muscular paralysis, or ligamentous weakness from long standing or traumatism. Flat-foot is a sequence of rickets and infantile paralysis. Flat-foot is recognized by painting the sole of the foot with a colored fluid and causing the patient to stand upon a piece of paper, and noting whether an impression of the entire sole is left upon the paper.

Club-foot or talipes is a permanent fixation of the foot in deformity. In *talipes equinus* the heel is drawn up in such a manner that the patient walks upon the ball of the foot or the toes. In *talipes varus* the foot is inverted, the patient walking upon its outer border. In *talipes valgus* the foot is everted and the patient walks upon the inner border of the foot.

Enlargement of the foot with more or less distortion occurs in acromegaly, myxedema, and pulmonary osteoarthropathy.

Erythromelalgia.—In this condition the sole of the foot is very red and the seat of burning pain, which is made worse by walking and is relieved by elevating the limb.

THE LEG

Bowing of the tibiæ is most commonly due to rickets, but may also be noted in connection with osteitis deformans, mollities ossium, and cretinism.

Nodes.—Red, shining nodes situated over the tibiæ, which are very painful upon pressure are indicative of erythema nodosum, which is more frequently encountered here than in any other locality.

Deep nodular swellings, situated upon the tibia are due to syphilitic periostitis, while painless, noninflammatory indurated areas distributed over the leg may be gummata.

Leg Ulcers may be due to varicose veins, but are often due to tertiary syphilis, especially if there are multiple annular ulcers situated nearer the knee than the ankle.

Swelling of the Calves in children, associated with loss of muscular power and difficulty in rising to the erect posture, is indicative of pseudohypertrophic muscular palsy.

Atrophy of the muscles clothing the anterior and outer aspects of the leg is a sign of progressive muscular atrophy.

Varicose Veins.—Varicosities of the veins of the leg are indicative in some instances of prolonged standing, or the pressure of a pregnant uterus or tumor within the abdomen upon the vessels returning blood from the lower extremity.

an enormous size, due to chronic inflammation of the synovial lining of the joint, which later progresses to the bone itself. The enlargement of the joint is always considerable and may become enormous. Early in the case the enlargement is due to effusion in the joint, but later it is produced by true osseous overgrowth. Pain is slight or is entirely absent. Usually affecting the knee-joint, the condition may involve the hip joint, and less commonly the smaller articulations.

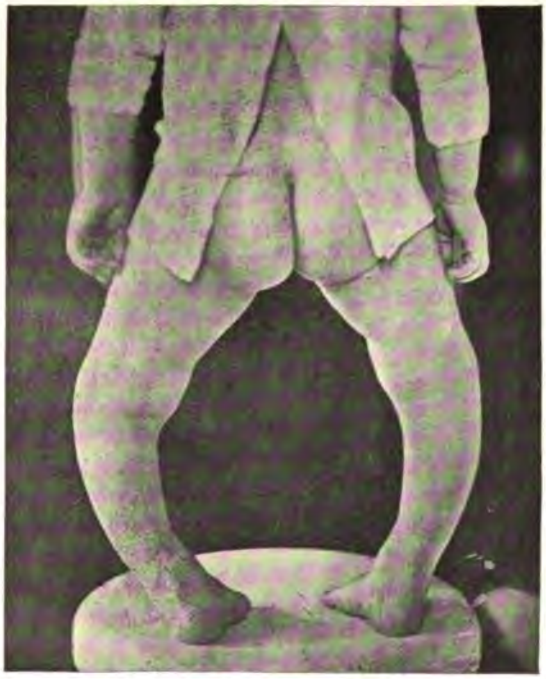


Fig. 309.—Showing extreme case of bowlegs. (From Woolley.)

Housemaid's knee, produced by chronic bursitis of the prepatellar bursa as a consequence of persistent pressure upon the bursa incident to occupation, is characterized by effusion into the knee-joint, the effusion pushing the patella upward before it. Fluctuation can sometimes be obtained.

THE THIGH

Edema of the thigh, affecting the leg and foot as well, possesses definite significance depending upon whether it is unilateral or guinal adenitis of long standing is suggestive of tuberculous disease of the hip or knee, or Hodgkin's disease. In malignant disease of the genitalia there is early inguinal adenitis.

Swelling in Scarpa's Triangle.—A swelling in this portion of the thigh may be due to femoral hernia, or psoas abscess, the latter always occupying a position external to the femoral vessels.

Osteitis Deformans (Paget's Disease).—Osteitis deformans produces bowing of the bones of the thighs, with a consequent diminution of the stature. The head in this disease is characteristically deformed, and the contour of the thorax and abdomen is altered.

Osteomalacia, in its evolution is characterized by bowing of the bones of the lower extremity, produced by softening and rarefaction of the osseous structures peculiar to this disease.

Rickets.—In advanced rickets there is usually notable bowing of the bones of the lower extremities, leading to "bowlegs" or "knock-knees." In addition there are symmetrical swellings at the epiphyses of the long bones.

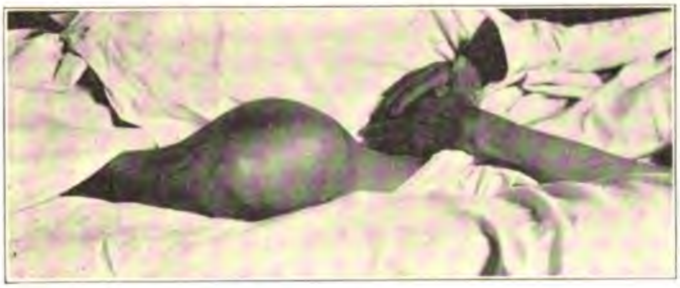


Fig. 311.—Osteosarcoma of femur.

Pulmonary Osteoarthropathy.—In this disease the extremities of the long bones of the lower extremity, particularly of the tibiæ, participate in the chronic enlargement which characterizes the disease.

Phlegmasia alba dolens, resulting from thrombosis of the femoral vein, produces swelling and edema of the thigh, with marked tenderness upon manipulation. The usual cause is puerperal sepsis, but this condition is also a not infrequent complication of typhoid fever.

Paralysis.—Paralysis of one leg if spastic is usually a part of a hemiplegia, but may rarely be due to a cortical lesion involving the leg center. Flaccid paralysis of one leg is the result of pres-

PART IV

EXAMINATION OF THE NERVOUS SYSTEM

SECTION I

MOTOR AND SENSORY PHENOMENA

CHAPTER XXXIII

STATION, GAIT, AND MUSCULAR POWER—TREMOR

Introduction.—The sources of the nervous impulses which initiate muscular movements reside in certain specialized cells of the cerebral cortex lying anterior to the fissure of Rolando, in the nuclei of the cranial nerves at the base of the brain, and in the anterior horns of the spinal cord. The experimental work of Hughlings Jackson, Hitzig, Ferrier, and Horsley has demonstrated that the motor path from the cerebral cortex to the voluntary muscles comprises two segments, or neurones; namely, the *upper motor neurone,* extending from the cerebral cortex to the anterior cornual cells, and forming synapses with the cells of certain of the nuclei of origin of the cranial nerves; and the *lower motor neurone,* which extends from the anterior cornual cells to the muscle in question.

The axis cylinders of the *upper motor neurone,* arising from cells of the cerebral cortex in the motor area pass downward into the white substance of the brain to form the corona radiata. They are collected into a compact bundle of fibers which traverse the internal capsule between the basal ganglia, constituting the genu and anterior two-thirds of the posterior limb of this structure. Emerging from the internal capsule, the upper motor neurone enters the crus cerebri, some fibers at this point crossing to the opposite side to form synapses with cells of the nucleus of origin of the oculomotor nerve. The upper neurone traverses the crus and pons, distributing fibers to all of the motor cranial nerves of the opposite side and a few fibers to the same nerves on the same side, and enters the anterior portion of the medulla oblongata to form the pyramid. In the medulla the greater number of the fibers constituting the upper motor neurone cross to the opposite side, forming the decussation of the pyramid. These fibers enter the lateral portion of the spinal cord as the crossed pyramidal tract, while the smaller number of fibers, which did not cross at the decussation, pass down the anterior portion of the cord as the direct or uncrossed pyramidal tract. The fibers of the crossed and direct pyramidal tracts terminate at various levels of the cord by forming synapses with the anterior cornual cells, the direct pyramidal fibers crossing in the anterior white commissure before forming this junction. Thus the upper motor neurone terminates by effecting a junction with the cells of origin of the lower motor neurone. It is to be noted that impulses arising in the cerebral cortex are all transmitted to the opposite side of the spinal cord by the upper motor neurone.

The axis cylinders of the *lower motor neurone* arise in the anterior cornual cells and emerge as the anterior spinal nerve roots to form the peripheral nerves which supply muscles on the same side of the body. They do not cross.

The sensory conducting system comprises three neurones. The *first sensory neurone* is derived from the ganglia upon the posterior nerve roots, the axis cylinders of which divide in a T-shaped manner, the longer division going to the peripheral sensory nerve, while the shorter branch enters the posterior horn of the spinal cord and divides into a long ascending and a short descending branch. The longer, ascending branches from this source ascend in the posterior columns of the cord to terminate in cells of the gray matter of the same side of the cord or to ascend to the nucleus gracilis and nucleus cuneatus of the medulla.

The *second sensory neurone* arises from the medullary cells or the medullary nuclei, form the arcuate fibers, and terminate in synapses about the cells of the median and lateral nuclei of the optic thalamus of the opposite side.

The *third sensory neurone* takes origin from the nuclei of the optic thalamus and terminates in the sensory areas of the cerebral cortex.

Gross lesions involving the integrity of the upper motor neurone in any portion of its course from the cerebral cortex to the anterior horns of the cord produce spastic paralysis of definite portions of the muscular system; since the regulating or governing impulses descending from the cerebral cortex are in abeyance and the constant tonic impulses from the anterior cornual cells are uncontrolled. Lesions of the lower motor neurone, on the contrary, produce flaccid paralysis, with atrophy of the muscle, as trophic impulses have their origin in the anterior cornual cells.

The Station.—The station is the attitude of the patient when standing at ease in the erect posture. In testing the station the patient should be directed to stand with the feet closely approximated, and the test should be made first with the eyes open and then with the eyes closed. A normal person while undergoing this examination will frequently sway slightly from side to side, and in cases of muscular weakness, either from exhausting disease or from neurasthenia, the swaying is more marked. But when the swaying movement becomes so extreme that the patient is in danger of falling if not supported, the station becomes pathologic. Thus in tabes dorsalis the patient with feet closely approximated and the eyes closed sways excessively and if not supported is apt to fall (Romberg's sign).

The Gait.—In many nervous diseases the gait is characteristic and gives at once a clue to the correct diagnosis. In observing the gait of a patient who is suffering with an organic nervous disease the clothing should be removed from the lower extremities so that the phenomena attending locomotion may be clearly observed.

The Spastic Gait.—In spastic diplegia due to lesions in the lateral pyramidal tracts the lower limbs are stiff owing to an inability to bend the knees, so that the patient progresses by means of short steps, the toes scraping along the floor. The toes of the shoes are worn excessively. The presence of a marked ankle clonus on both sides communicates a general tremulousness to the entire carriage of the patient, who is apt to stumble over slight obstacles and fall.

The Hemiplegic Gait.—The hemiplegic gait is merely a unilateral spastic gait, the spastic limb during progression describing an arc of a circle while the sound limb supports the weight of the body. In spastic cerebral paraplegia, or double hemiplegia, both limbs describe the arc of a circle during progression, each foot in turn being swung outward and planted in front of the other with the production of the cross-legged or reich's ataxia, and in tumor of the posterior columns of the cord. The patient walks on a very broad base, swaying from side to side. The foot in progression is raised suddenly from the floor, is thrown forcibly forward, and thrown forcibly down "in flail-like fashion," the heel usually striking the floor first. The patient keeps the eyes fixed steadily upon the floor before him in the effort to guide his onward course. He is unable to suddenly stop or start on command or to turn suddenly and retrace his course. Similar ataxia in the upper limbs is demonstrable in the inability of the patient to touch the finger-tip to the nose, or to accurately approximate the finger-tips with the arms before the body.

The Festinating Gait.—In paralysis agitans the patient moves forward with the body inclined somewhat forward, advancing with short, shuffling steps which become progressively faster as he crosses the room. When ordered to turn, the entire body is turned *en masse.* This type of locomotion constitutes the so-called *propulsion. Retropulsion* may often be elicited in these patients. If the patient is quickly pulled backward, and, indeed sometimes on merely looking upward, he tends to run backward with short, shuffling steps, although the body is invariably inclined forward.

The Cerebellar or Vertiginous Gait.—In cerebellar disease associated with severe vertigo the patient progresses in a very irregular course, often lurching from side to side. Quite frequently the patient exhibits a tendency to reel in a fixed direction, forward, backward, or to one side. This type of progression occurs with unilateral cerebellar lesions. In unilateral cerebellar tumors the head is not infrequently inclined toward opposite side, while the face is turned slightly toward the side of the lesion.

Muscular Power.—A rough estimate of the muscular power may be made by the "resistance method," the patient being directed to perform the function of a given muscle, while the examiner endeavors to resist the movement and gauges the amount of power required in the effort. Variations in muscular power range from simple weakness to complete loss of power or paralysis. Paralysis may be complete or partial, in which latter event it is termed paresis. Paralysis may be *spastic,* when the paralyzed limb is rigid and the muscles unyielding to passive movement, or *flaccid,* when the muscles are soft and pliable. Paralysis may affect one limb, when the condition is termed *monoplegia*: it

CHAPTER XXXIV

SENSORY PHENOMENA—THE REFLEXES

An accurate sensory examination requires the free and full cooperation of the subject of the examination and a refined technic upon the part of the examiner. The area under examination should be freely exposed and the examination should be conducted in a chamber which is free from noise and in which the temperature is constant and is neither uncomfortably elevated nor lowered. The sensory examination should not be unduly prolonged; but when any disorder of sensation is detected, its exact limits should be determined. In this connection it is important that the examiner should refrain from graphically charting the limits of the area of dysesthesia upon the skin, as such practice serves as a fertile source of suggestion to the subject of the examination.

The course of the afferent sensory pathway has been outlined in a previous section; and from its course it is evident that a sensory disturbance may be due to a lesion involving the integrity of a peripheral nerve trunk, involving one or more of the tracts of the spinal cord, or involving the brain stem or the cerebral cortex. The area and distribution of the sensory disturbance will in each event serve to localize the site of the causative lesion. Disturbances of general sensation comprise disorders in the sense of touch, pressure sense, the senses of temperature and pain, and of muscular sense, which comprises the perception of active and passive movements. Disturbances of special sense are treated in the section dealing with the several cranial nerves.

Tactile Sensation.—The acuity of tactile sensibility is tested by gently touching the cutaneous surface in various regions with a feather, a camel's-hair brush, or with a twisted wick of cotton, while the subject's eyes are bandaged. Tactile sensation is also investigated by detecting the minimal distances at which the two points of a compass may be appreciated when simultaneously placed upon the skin of the subject.

The normal subject can state the precise instant at which the integument is touched and, in a general way, the nature of the fabric with which it is in contact. The readiness with which the two compass points are recognized varies widely in different regions of the body, being most sensitive upon the tip of the tongue, where they are recognized when separated by only one millimeter, and least readily detected upon the back, arm, and thigh, where they are only recognized when separated by a space of from 60 to 80 millimeters. While the readiness with which tactile sensations are registered is a reliable index to the integrity of the sensory pathway, allowance must be made in certain instances of delayed transmission for the degree of natural intelligence of the subject of the examination.

Moderate impairment of tactile sensibility constitutes hypesthesia, indicating in the majority of cases a compression or partial lesion of the sensory pathway. An abnormally acute perception of tactile sensation constitutes hyperesthesia, which frequently indicates a functional irritability of the sensory pathway. A complete abolition of tactile sensibility over a zone of the body constitutes anesthesia. A circumscribed anesthesia is likely to prove of peripheral origin, whereas an extensive zone, embracing one-half of the body or the entire body below a definite level is of central origin.

In the presence of a complete transverse lesion of the spinal cord there is anesthesia and motor palsy which is bilateral below the level of the lesion. In the presence of a hemisection of the cord or of a lesion involving only one lateral half of the cord, there ensues the phenomenon which is designated Brown-Séquard paralysis, in which there is complete motor palsy upon the corresponding side below the level of the lesion, with complete anesthesia and partial loss of motor power upon the side opposite to the cord lesion.

Pressure Sense.—The pressure sense is investigated by noting the ability of the subject to appreciate minor variations in pressure, when cubes of uniform size but of varying weight are placed upon the surface under examination. During this examination muscular sensation is to be eliminated by placing the limb upon a firm, unyielding surface; and temperature sense must be excluded, as extremes of temperature have a tendency to impair the nicety of the pressure sense perception. Variations in pressure sense
which possess the same significance as do similar variat
tactile sensation, are not relied upon as much as are the l
neurologic examinations.

Sense of Temperature.—The entire cutaneous surface
plied with specific "heat spots" and "cold spots," w
y nerve endings for the appreciation of these

of temperature sense. Hence the power of discriminating variations in temperature may be retained, while tactile sensation is temporarily or permanently abolished. Compression of the ulnar nerve, which causes a marked diminution of tactile sensibility over the distribution of the nerve, does not involve the temperature perception in this area. As a general rule, the portions of the body which are habitually clothed are more sensitive to thermic variations than are the exposed portions of the integument.

The cutaneous perception of temperature is most conveniently tested by the application to the area under examination alternately of test tubes containing water at temperatures considerably above and below the body temperature. In the presence of organic disease of the spinal cord, as syringomyelia and in the presence of lesions of the medulla and pons, as hemorrhage, tumor, or softening, the perception of temperature is impaired (thermohypesthesia). Similarly destructive lesions in these regions result in total abolition of the perception of changes of temperature, constituting thermoanesthesia; whereas functional irritability of the tract of Gowers may result in an exaggerated perception of variations in temperature, constituting thermohyperesthesia.

Sense of Pain.—While it is true that any form of appreciable sensation when sufficiently magnified and intensified may assume such proportions as to render its perception painful, it is yet generally agreed that there are specialized "pain spots" distributed universally over the cutaneous surface, which are supplied by special nerve endings; and that the sensation of pain when pronounced is not invariably due to overstimulation of the fibers having to do with tactile or temperature sensation, but to hyperstimulation of these specific pain centers in the integument.

The perception of the sense of pain is investigated by pricking the integument of the area under examination with a sharpened quill or with an ordinary pin or needle. In the presence of central cord lesions which are incomplete, in dorsal sclerosis of the cord and in lesions of peripheral nerves, the perception of painful stimuli may be delayed and impaired, constituting hypalgesia, or may be totally abolished in the area under investigation, constituting analgesia. Similarly in the presence of functional nervous states the examiner is apt to encounter an exaggerated perception of painful stimuli, constituting in this event hyperalgesia.

Different forms of perverted sensation may be encountered, which are grouped under the term parasthesiæ. These perversions are principally subjective in character and are not modified or influenced by objective examination of the subject. They may assume the character of the crawling of insects (formication); they may take the form of alternate flashes of heat and of cold; or the subject may complain of transient numbness. Occasionally upon painful stimulation of one extremity there is experienced coincidentally a painful sensation in a symmetrical distribution of the opposite limb, constituting in this instance, allocheiria.

Muscular Sense.—Muscular sensation is the peculiar sense by means of which judgments are formed as to the weight of articles which are lifted, by which the patient is aware of the position of certain portions of the body without the aid of the eyes, and by which he is enabled to maintain the standing posture without conscious effort.

The muscular sense may be examined by directing the subject, with the eyes lightly bandaged, to place the finger upon a certain designated portion of the body, as for instance, the tip of the nose; it is also tested by directing the patient to stand upright with the feet closely approximated and with closed eyes. Thus, in organic disease of the nervous system the disturbance of the muscular sense, with coincident tactile sensory disturbance, is responsible for Romberg's sign.

The muscular sense is also investigated by noting the perception of active and passive movements of the limbs. Thus, the subject is directed to perform various movements with the limbs, such as describing a semicircle on the floor with the toe, or touching the knee with the ankle of the opposite limb. In testing the perception of passive movements, the limb of the patient is slowly moved, while the eyes are lightly bandaged, and he is asked to indicate by pointing the range of movement and the new position of the limb.

Stereognostic Sense.—Stereognostic sensibility is the faculty by which objects placed in the hand are recognized by their palpable shape and consistence. An abolition of this sense (astereognosis) is frequently indicative of a lesion involving the superior parietal lobule of the brain.

Reflexes.—*The Reflex Arc.*—The simplest form of reflex arc comprises an afferent or sensory neurone, which conducts impulses from the periphery of the body to an intermediate cell station situated in the gray matter of the spinal cord, and an efferent motor neurone, over which the intermediate cell station or medullary center discharges impulses in response to the sensory stimulus which is conveyed to it by way of the afferent sensory neurone. The entire sequence of changes which ensues upon adequate stimulation of the receptor of the afferent sensory neurone constitutes the reflex act.

In the case of the spinal reflexes the afferent neurone is represented by the peripheral spinal sensory nerve with its root ganglion, the intermediate cell station by the cells of the gray matter of the spinal cord, and the efferent neurone by the motor nerve arising from the cells of the anterior horn of the cord. The shorter branch of the dorsal nerve root upon entering the posterior horn of the spinal cord gives off collaterals which pass immediately to form synapses with the cells of the anterior horns of the same side of the spinal cord, a reaction in this instance constituting a homolateral spinal reflex. However, the spinal nerve root also furnishes collateral branches which pass by way of the anterior white commissure to form synapses with the anterior cornual cells of the opposite side of the cord, resulting in contralateral reflexes. Also certain collateral branches form synapses with motor cells in higher and lower levels of the spinal cord. A much more complicated reflex is represented by the collaterals which establish connections with the cells of origin of the columns of Goll and Burdach, by which they are relayed to the gracile and cuneate nuclei of the medulla, thence by the arcuate fibers to the opposite optic thalamus, and thence by the third sensory neurone to the sensory area of the cerebral cortex. In this situation an intermediate cell station in the form of association bundles is established, the efferent motor neurone of this extensive reflex arc being represented by the pyramidal tracts, the reflex action resulting in consciousness and in volitional muscular movement. Another very complex reflex arc is formed by the dorsal root fibers which form synapses with the cells of Clarke's column, whence the impulse is relayed by the ascending spino-

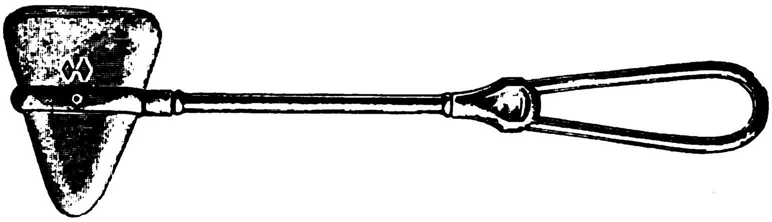


Fig. 314.—Percussion hammer.

cerebellar tract of Flechsig to the cerebellar cortex and dentate nucleus, thence to the red nucleus of the midbrain, and thence by the rubrospinal tract to the anterior cornual cells, the reflex act in this instance having for its expression the regulation of muscle tone and the synergic action of different muscles in the maintenance of equilibrium.

Simple spinal reflexes occur without any intervention upon the part of the cerebrum, the various spinal segments acting independently; but there are governing fibers descending in the pyramidal tracts from the cerebral cortex, which modify and may volitionally inhibit the spinal reflexes.

Reflexes are variously classified as tonic and clonic, as superficial reflexes and as deep reflexes. The principal superficial reflexes are the plantar, cremasteric, and abdominal reflexes; while the principal deep reflexes comprise the patellar and achilles tendon reflexes, the jaw-jerk, the ankle clonus and the patellar clonus.

Abdominal Reflex.—The abdominal reflex consists in a contraction of the underlying rectus muscle when the integument is stimulated, as for instance, by gently scratching with a sharp instrument. This reflex should be elicited upon either side of the abdomen both above and below the level of the umbilicus, in order to demonstrate the independence of the upper portion of the rectus, which is supplied by the eighth and ninth intercostal nerves; and the lower portion of the rectus, which is supplied by the tenth and eleventh intercostal nerves. In each case, the umbilicus is observed to be drawn toward the side of the stimulation in the presence of an intact reflex. It may readily be demonstrated that in the case of the normal subject the umbilicus is not displaced upon changing from the dorsal recumbent to the sitting posture. But in the presence of paralysis of the lower portion of the rectus, upon the change of posture the umbilicus is elevated, thus differentiating the lesion of the tenth and eleventh intercostals from the integrity of the eighth and ninth intercostal nerves above the umbilicus.

Cremasteric Reflex.—Upon stimulation of the integument upon the inner side of the thigh the testicle of the corresponding side is elevated. The segments which preside over the cremasteric reflex comprise the first and second lumbar segments.

Plantar Reflex.—In the normal subject, when the sole of the foot is stroked with a probe or a sharp instrument, plantar flexion of all of the toes ensues as the reflex response. In the presence of a lesion of the upper motor neurone, instead of the uniform plantar flexion of the toes, the great toe is extended while the remaining toes are flexed (Babinski's sign). In eliciting the plantar reflex the outer portion of the sole should be stroked in a direction from before, backward toward the heel. Subjects yielding Babinski's sign usually also yield upon proper stimulation the reflexes of Oppenheim and of Gordon.

Oppenheim's Reflex.—Oppenheim's reflex is elicited by passing the thumb or the handle of the percussion hammer downward along the internal border of the tibia, at the same time exerting pressure upon the soft tissues, whereupon extension of the great toe with plantar flexion of the remaining digits ensues. The significance of Oppenheim's reflex is entirely identical with that of the pathologic plantar reflex.

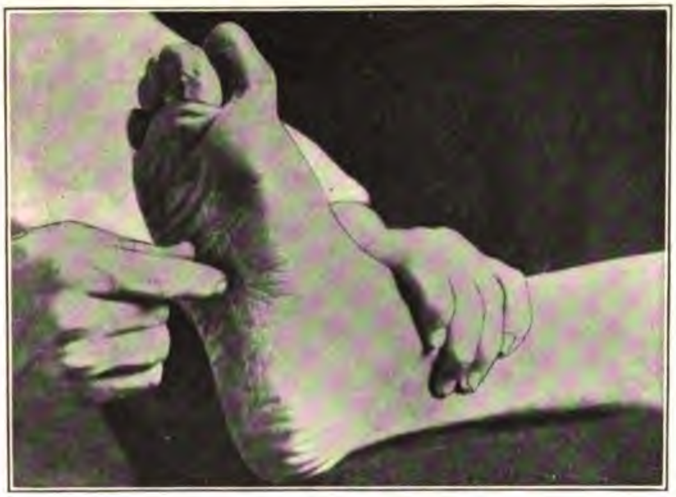


Fig. 315.—Elicitation of Babinski's sign.

Gordon's Reflex.—If, instead of proceeding as in the case of Oppenheim's reflex, the examiner merely grasps the muscles of the calf of the leg and exerts deep pressure, in the presence of a lesion of the upper motor neurone, extension of the great toe ensues.

The Patellar Tendon Reflex (Knee-jerk).—If the patient is in the sitting posture, to elicit the knee-jerk the leg is flexed upon the thigh at a right angle, while the patellar tendon is struck a rapid light blow with the percussion hammer. If the patient is bedridden, the reflex may be elicited by raising the leg from the bed by means of a hand placed beneath the knee joint while the blow is delivered.

During the examination every effort should be made to distract the attention of the patient from the procedure in order to prevent cerebral inhibition of a normal reflex. If the subject is very selfconscious and the reflex consequently is elicited with difficulty, Jendrassik's reinforcement may be resorted to. The patient is directed to lock the hands and to pull, meanwhile keeping his eyes fixed upon the ceiling. The reflex arc of the patellar tendon reflex comprises the second, third, and fourth lumbar segments.

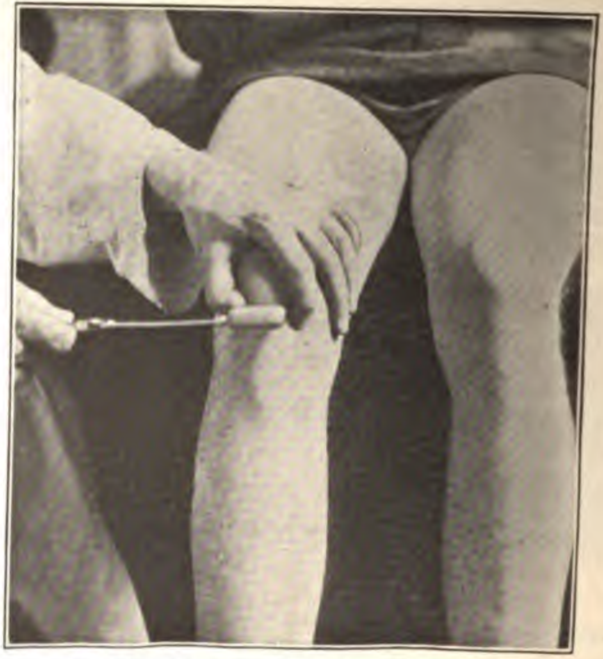


Fig. 316.—Elicitation of patellar tendon reflex.

Exaggeration of the patellar tendon reflex is indicative of disease between the level of the reflex arc and the cerebral cortex, whereby the governing impulses from the cerebrum are interrupted. Such a condition arises in spastic spinal paraplegia, amyotrophic lateral sclerosis, cerebral hemorrhage, and disseminated sclerosis.

Abolition of the patellar tendon reflex is indicative of a break in the reflex arc due to disease of the sensory neurone, posterior root zone, or anterior root cells. Such lesions arise during the progress of tabes dorsalis, anterior poliomyelitis, peripheral neuritis, and trauma to the cord at the level of the reflex arc.

The Tendo Achillis Reflex.—This reflex may be elicited by directing the patient to kneel upon a chair, and, rendering the tendon taut by moderate dorsal flexion of the foot, the tendo Achillis is struck sharply, whereupon normally a sudden extension of the foot is produced. The reflex arc concerned in this reflex comprises the first and second sacral segments; and the significance of variations in the response is identical with those detailed under the knee-jerk.

The Jaw-Jerk.—In the presence of amyotrophic lateral sclerosis upon striking the point of the chin with the percussion hammer while the mouth is half opened the jaw-jerk is elicited. This reflex is distinctly pathologic and is not present in the normal subject. The reflex arc is represented by branches of the trigeminal nerve.

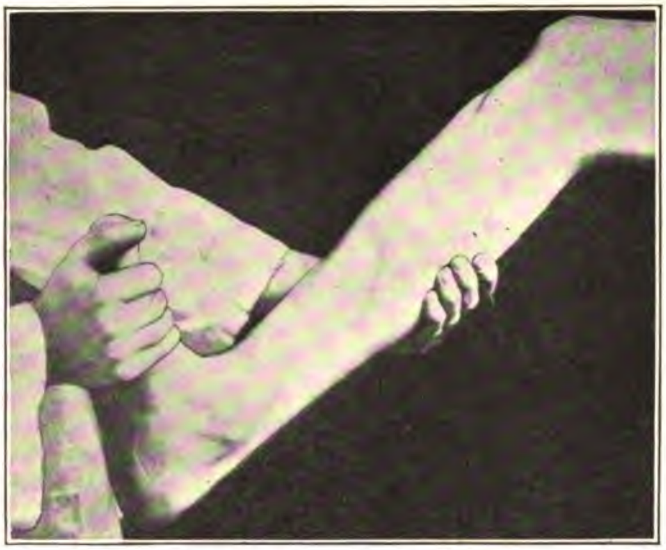


Fig. 317.—Elicitation of ankle clonus.

Ankle Clonus.—In eliciting this clonus the examiner grasps the calf of the leg in the palm of the left hand, while with his right hand he exerts pressure upon the fore part of the sole of the foot, thus maintaining the foot in a position of dorsal flexion. In the presence of disease of the upper motor neurone, as in disseminated sclerosis, cerebral hemorrhage, or spastic paraplegia, a series of regularly rhythmical contractions of the calf muscles

CHAPTER XXXV

THE CRANIAL NERVES

The twelve cranial nerves are paired nerves, resembling in this respect the spinal nerves. The first two cranial nerves, however, the olfactory and optic, differ so markedly in their anatomic and physiologic features from the other cranial nerves as to have been compared to accessory lobes of the brain. The centers of the cranial nerves lie in a mass of gray matter along the floor of the fourth ventricle, the aqueduct of Sylvius and the floor of the third ventricle, representing an upward continuation of the central gray matter of the spinal cord.

Lesions involving the cranial nerves may be situated in the cerebral cortex or the fibers descending from the cortical cells to the deep origin of the cranial nerves (supranuclear lesions), may involve the nucleus alone (nuclear lesions), or may involve only the peripheral portion of the nerve (infranuclear lesions). While supranuclear and infranuclear lesions not infrequently manifest themselves in derangements of a single cranial nerve, the nuclei of origin of these nerves are so closely aggregated beneath the floor of the fourth ventricle and sylvian aqueduct that a lesion in this situation usually involves the nuclei of several cranial nerves, with the consequent production of more general manifestations.

THE OLFACTORY NERVE

The center for the olfactory nerve is probably situated in the uncinate and hippocampal gyri, with communicating fibers to the cerebral cortex, optic thalamus, and internal capsule. The terminal branches of distribution of the nerve are distributed to the superior turbinated bodies and the upper portion of the septum, whence they pass upward to the dilated anterior extremities of the olfactory tracts, the olfactory bulbs.

The integrity of the olfactory nerve is tested with familiar odorous substances, such as the oils of peppermint or cloves, cologne water or cinnamon. Ammonia or acetic acid should not be employed, as they are known to affect the trigeminal nerve. In and the left tract supplying similar fibers to the left half of each retina. Lesions involving different portions of these tracts produce characteristic lesions which aid in localizing the individual lesion.

The light fibers, the fibers of the optic nerve and retina which react to light stimuli, arise in the retina, whence they pass backward in the optic nerve, undergoing partial decussation at the chiasm, and proceed along the optic tracts to the external geniculate bodies and corpora quadrigemina, whence they pass to the oculomotor nucleus beneath the floor of the aqueduct of Sylvius by way of the fasciculus sublongitudinalis. Thus the reflex arc of the light reflex is composed of an afferent limb, a substation in the midbrain, and an efferent limb, which will be considered in detail under the examination of the third cranial nerve.

Vision.—The acuity of vision normally is tested with the ordinary Snellen Test Type.

Amblyopia.—Amblyopia, dimness of vision, which is not due to errors of refraction, may result from the excessive use of tobacco or alcohol. Amblyopia may also arise during diabetes mellitus, or it may signify impending uremia in a nephritic patient. The ingestion of certain drugs, as quinine or the salicylates, may induce amblyopia.

Hemeralopia (Day-Blindness).—Hemeralopia, a condition in which the vision is impaired during the day, but improves on dark days or at night, is often part and parcel of tobacco amblyopia. It may also signify chronic optic neuritis from intracranial causes or intoxications, or chronic retinitis from a similar cause.

Nyctalopia (Night-Blindness).—Nyctalopia, characterized by imperfect vision in subdued light, is often the result of frequently repeated exposures to strong illumination. In other instances it is a congenital defect of the visual apparatus.

Color Vision (Color-Blindness).—The inability to differentiate between differences in the gradation of colored fabrics is in most instances an inherited defect. Acquired color-blindness occurs, however, as the result of toxic amblyopia, optic neuritis, or as a rare result of trauma to the cranium. In testing for color-blindness, the Holmgren or Thomson test should be employed.

Holmgren Test.—In applying this test the patient is given a skein of wool of a light-pink color and directed to select from a mass of similar skeins of various colors and shades of colors all those which nearly match the color of the selected test skein.

If the color vision is impaired, skeins of varying colors, gray, green, pink, and brown, will be selected indiscriminately. If the subject fails on the pink skein, a pure green skein is selected for a control.

Thomson's Test.—In this test a stick to which numerous bundles of yarn of various colors are attached is employed. The colors have corresponding numbers, the odd numbers being green and the even numbers corresponding to the confusing colors. The color vision is tested with a pale green test skein, the patient being required to match it with ten tints on the rod. The selection of skeins with even numbers reveals the patient's inability to discriminate between the different shades and colors. A control test should be made with red and old-rose skeins as test colors.

Field of Vision.—The dimensions of the field of vision in each eye is best determined by a perimeter; but as this instrument is usually not available, other methods of testing the field of vision must be employed. A rough but sufficiently accurate estimation of the size of the field of vision may be made by the following simple procedure.

The patient is seated in a straight chair with his back toward the source of illumination, the examiner occupying a chair facing the patient, and approximately three feet from him. In testing the left eye, the right eye of the patient is covered with a bandage; the examiner closes his right eye, at the same time fixing his left eye upon the pupil of the left eye of the patient. The examiner, beginning well beyond the limits of vision for both patient and himself, slowly moves his hand inward until the patient first sees the finger-tips. This maneuver is repeated in all the meridians of the visual field; and if the finger-tips become visible to the patient at the same instant they are apprehended by an examiner with a normal visual field, the patient's visual field is of normal extent; that is, is not contracted. If, on the contrary, the hand of the examiner must be brought nearer the visual axis than is required for the normal examiner, the visual field of the patient is contracted.

Contractions of the visual field may be *concentric* or *irregular.* Concentric contraction is noted in many cases of hysteria, and also in glaucoma. Irregular or asymmetric contractions, represented by scotomata and hemianopia possess a varied significance.

Scotomata are to be detected by passing small pieces of white and colored cardboard ac f vision while the patient fixes the eye under examination upon a designated objective point. Under these circumstances the patient is directed to state the point in the progress of the cardboard at which it becomes temporarily invisible. It is to be remembered that there is a physiologic scotoma for light and color, corresponding to the blind spot of Mariotte, which must be eliminated in ocular examinations. An *absolute scotoma,* betrayed by the inability of the patient to recognize in the scotomatous field a white cardboard or light stimuli, is significant of grave destructive lesions, as optic neuritis or a lesion involving some portion of the optic tract. A *relative or color scotoma,* revealed by the inability of the subject to appreciate red and green cards in certain portions of the visual field, is usually the result of the excessive use of tobacco or alcohol, and gives a distinctly better prognosis than do the absolute scotomata.

Hemianopia, obliteration or darkening of one-half of the visual field, is tested for clinically by the maneuver used for determining variations in the extent of the visual field; which, in the presence of hemianopia reveals a darkening of one-half of the visual field.

Hemianopia may be horizontal or vertical, homonymous or heteronymous, bitemporal, binasal or mixed, as the case may prove.

The significance of hemianopia is a lesion involving the optic nerves, optic chiasm, or optic tract; and the site of the intracranial lesion is determined by the distribution of the hemianopic changes. In homonymous hemianopia the corresponding halves of the visual fields are obliterated; as, for instance, the temporal half of the right retina and the nasal half of the left retina. Such an ocular finding constitutes left lateral homonymous hemianopia, the significance of which is a lesion involving the right optic tract alone. Similarly a bitemporal hemianopia signifies a lesion involving the central portion of the chiasm, whereas a binasal hemianopia is produced by lesions at both extremities of the chiasm, but sparing the central portion of this structure, a condition which rarely occurs. Transitory hemianopia sometimes occurs with hysteria and migraine without anatomic change in the tract.

Wernicke's Pupillary Reaction.—If, in a case of hemianopia, with the patient seated in a darkened room, a thin ray of light from an ophthalmoscopic mirror is projected into the orbit upon the hemianopic retinal area at an angle of 40 to 60 degrees from the visual axis, myosis may or may not result. In hemianopias ciculus sublongitudinalis, which forms the connecting link between the corpora quadrigemina and the third nerve nucleus.

Consensual Light Reflex.—If during the examination for the light reflex in one pupil, the pupil of the opposite eye is observed, while shaded and protected from the light stimuli applied to the opposite retina, it will be observed to react along with the pupil of the exposed eye. This phenomenon constitutes the consensual light reflex, and is due to the transmission of an impulse across the fibers which connect the two third nerve nuclei.

Reaction to Accommodation.—When the range of vision is suddenly transferred from a distant objective point to an object near at hand, the pupils will be observed to contract and the eyes to converge, the reaction to accommodation. This reaction may be quickly tested by directing the patient to fix the gaze on a distant portion of the room, and then quickly to transfer the gaze to the finger of the examiner held near the face of the patient. Abolition of this reflex is due to third nerve paralysis.

Argyll-Robertson Pupil.—Abolition of the light reflex in one or both eyes with retention of the reaction to accommodation constitutes the Argyll-Robertson pupil, which is occasionally found in disseminated sclerosis, and very frequently in tabes dorsalis and paresis. Marina has shown this type of pupillary reaction to be caused by degenerative changes in the ciliary ganglion. In this pupil the pupillary margins are very frequently irregular, while the pupils are often somewhat myopic (spinal myosis) from disease of the cervical cord. Ultimately in tabes and paresis the pupil becomes immovably fixed, reacting neither to light nor to accommodation.

An opposite pupillary reaction, the pupil reacting to light, but failing to react to accommodation is often seen as a sequence of postdiphtheritic paralysis.

Hippus.—Rapid, rhythmic, clonic contractions of the sphincter pupillæ producing winking movements of the iris which are so gross as to be visible to the unaided eye (hippus) are frequently demonstrable in disseminated sclerosis, more rarely in hysteria, incipient acute meningitis, and epilepsy.

Pupillary Unrest.—This phenomenon, which is a normal physical finding, consisting of a regular narrowing and widening of the pupil, is so fine that it can only be demonstrated by means of the aid of a magnifying lens with the pupil brilliantly illuminated. Abolition of this normal pupillary unrest is one of the earliest signs of tabes dorsalis and paresis.

Myosis.—Contraction of the pupil may result from irritative or destructive lesions. Irritative myosis is noted in the early stages of cerebral hemorrhage, in incipient brain tumors before sufficient pressure has been exerted upon the third nerve to cause paralysis, and in early acute meningitis and encephalitis. Paralytic myosis is seen in tabes dorsalis, the late stages of tabes of the cervical cord, and syringomyelia of this portion of the cord, leading to destruction of the pupil-dilating fibers.

Mydriasis.—Irritative mydriasis is often due to irritation of the pupil-dilating center in the cervical cord from congestion, spinal meningitis, or tumor. Paralytic mydriasis may signify paralysis of the sphincter pupillæ, caused by disease of the third cranial nerve or ciliary ganglion, increased intracranial pressure from brain tumor, or glaucoma.

Strabismus (Squint).—In paralysis of one or more ocular muscles the normal axis of the eyeball deviates from its normal position, with the production of double vision or diplopia. A simple rule in the differentiation of the various ocular paralyses is that the affected eye is displaced by the unopposed antagonists to the side opposite to the usual traction of the paralyzed muscle, while the false image, the result of diplopia, is displaced in the direction of the line of traction of the paralyzed muscle (Purves Stewart).

Nystagmus.—Nystagmus is a rapid oscillation of the globe of the eye upon voluntary motion, usually in a horizontal direction, more rarely in a vertical direction, and very rarely it is rotary. It is a sign of value in disseminated sclerosis, epilepsy, chorea, brain tumor, tabes dorsalis, Friedreich's ataxia, and in some cases of chorea. Nystagmus may be the result of errors of refraction and may be noted in albinos. Miners are subject to a form of nystagmus, probably caused by the constant excursion of the eyes while working in the recumbent or stooping posture.

Aural nystagmus, which may be produced experimentally by syringing the membrana tympani with water either above or below the temperature of the body, is regarded by Bárány as the result of convection currents produced in the endolymph by the warming and cooling of the labyrinth. This "thermic nystagmus" is of value in testing the integrity of the vestibular nerve.

Conjugate Deviation.—This comprises a concomitant deviation of both eyes toward the right or left, its significance being a lesion in the cerebral cortex, corona radiata, or internal capsule, above the crossing of the [illegible] Thus, in cerebral hemorrhage the eyes are turned toward the side of the lesion and opposite to the side of the paralysis (Prevost's sign). In interpreting the sign it is to be remembered that the lateral movements of the eyes are governed by impulses arising in the cerebral cortex and passing by way of the corona radiata and internal capsule to the sixth nerve nucleus of the corresponding side, and thence across the posterior longitudinal fasciculus to the subdivision of the opposite oculomotor nerve nucleus which presides over the internal rectus muscle. Thus the conjugate lateral deviation of the eyes is caused by the simultaneous stimulation of the external rectus muscle on the side of the lesion and of the internal rectus muscle on the side opposite to the lesion, causing the patient to "look at his lesion."

Ptosis.—Ptosis of the upper eyelid is revealed by the inability of the patient to elevate the lid. It is due to a lesion of the oculomotor nerve or nucleus. Isolated paralysis of the fourth cranial nerve is very rarely encountered, as this nerve usually participates in the palsies of the third and sixth nerves. In the rare instances of simple trochlear paralysis there is inability to rotate the globe downward and outward. Cerebral syphilis is the usual cause of the paralysis.

Abducent Paralysis.—Isolated paralysis of the sixth cranial or abducent nerve is revealed by the inability of the patient to rotate the eyeball outward beyond the midpoint. Upon endeavoring to follow the finger of the examiner the external rotation of the globe is interrupted at this point.

TRIGEMINAL NERVE

The trigeminal nerve has an extensive origin from the floor of the fourth ventricle, beneath the aqueductus sylvii, and the cervical spinal cord as low as the second cervical nerve. The fifth cranial is a mixed nerve, containing both motor and sensory fibers. The fibers constituting the sensory trunk have developed upon them the Gasserian ganglion which rests in a small fossa upon the petrous portion of the temporal bone. The motor root of the nerve supplies the masseters, the temporals, pterygoids, internal and external, mylohyoid, anterior belly of the digastric, the levator and tensor palati and tympani; and the azygos uvulæ. The sensory trunk and Gasserian ganglion terminate in three trunks, the superior and inferior maxillary, and the ophthalmic, which distribute sensory fibers to the anterior two-thirds of the tongue, the mucous membrane of the buccal and nasal cavities, the salivary glands and teeth, the infraorbital and mandibular portions of the face, and the anterior portion of the scalp.

Motor Paralysis.—Motor paralysis of the fifth cranial nerve is tested for by palpating the masseter and temporal muscles while the patient is directed to clench the teeth. In unilateral paralysis there is loss of the prominence with which the muscles stand out on the normal side. The patient is then directed to open the mouth and protrude the lower jaw. In unilateral paralysis the jaw is deviated toward the paralyzed side by the action of the sound external pterygoid muscle.

Irritative lesions of the motor trunk or centers produces *trismus,* a mild form of tetanic spasm of the muscles of the lower jaw. Severe tetanic spasm of these muscles accompanies tetanus and strychnine poisoning.

Sensory Paralysis.—Sensory paralysis involving the inferior maxillary division of the nerve produces anesthesia of the infraorbital region, which is tested for by drawing lightly across the face a small pledget of cotton loosely rolled, or a camel's hair brush.

Implication of the sensory fibers and the Gasserian ganglion are recognized by the very painful spasm, tic douloureux.

In testing the sense of taste over the anterior two-thirds of the tongue the patient is directed to protrude the tongue and the examiner places on it various substances, such as quinine, sugar, salt, and citric acid, in powdered form. While the tongue is protruded the patient is required to point out on a printed card whether the sensation appreciated is sweet, sour, bitter, salty, or negative. The patient should not be allowed to make his decision after the tongue has been returned to the oral cavity as the flavors may be carried by the saliva to the posterior portion of the tongue which is supplied by the glossopharyngeal nerve.

THE FACIAL NERVE

The nucleus, or origin, of the facial nerve lies in the lower portion of the pons near the medullary junction, the root fibers of the nerve emerging at the lower border of the pons just internal to the point of emergence of the auditory nerve. In company with the auditory nerve, the facial nerve enters the internal auditory meatus of the temporal bone, transverses the aqueductus fallopii of that bone, and emerges from the stylomastoid foramen. In the aqueductus fallopii the nerve receives the chorda tympani, which contains taste fibers from the anterior portion of the tongue. After emerging from the stylomastoid foramen the nerve divides

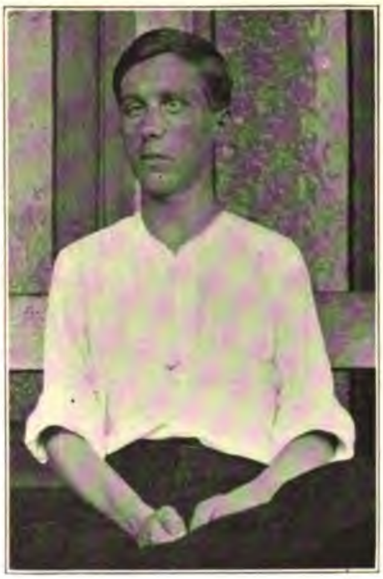


Fig. 318.—Facial paralysis.

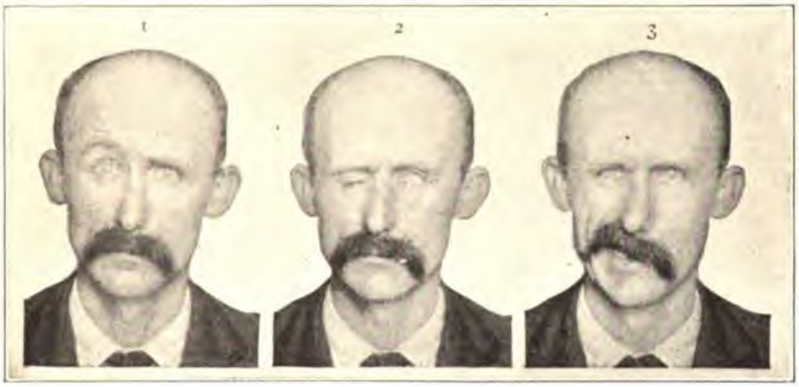


Fig. 319.—Facial paralysis. (Church.) *1*, bilateral attempt to raise eyebrows; *2*, bilateral attempt to close eyes; *3*, smiling. (From Eisendrath.)

into a number of diverging branches to supply the majority of the muscles of the head and face.

Facial Paralysis.—The facial nerve is purely a motor nerve, except for the fibers it receives from the chorda tympani, destructive the tuning fork should be applied to the midline of the forehead. Under these conditions, if the cause of the deafness lies in middle ear disease, the vibrations are most clearly audible in the diseased ear; whereas if it is a case of nerve deafness due to a lesion of the eighth nerve, the vibrations are audible only on the side of the sound ear.

Tinnitus.—Tinnitus aurium, or ringing in the ears, occurs with intracranial tumors and aneurysm, temporary obstruction of the eustachian tube during acute colds, and during disease of the labyrinth.

Vertigo.—Vertigo, or dizziness, when not of gastrointestinal origin, signifies a cerebral or intracranial lesion such as tumor or aneurysm acting upon the cerebellar centers of coordination or the afferent paths of the vestibular division of the auditory nerve. Tumors of the cerebellum are characterized by extreme vertigo and incoordination. Vertigo may be due to Meniere's disease or aural vertigo.

THE GLOSSOPHARYNGEAL NERVE

The glossopharyngeal nerve supplies sensory fibers to the mucous membrane of the pharynx and to the posterior third of the tongue. It is also the motor nerve of the middle constrictor of the pharynx and the stylopharyngeus.

Paralysis of the nerve is evinced by loss of taste sensation for the posterior third of the tongue and abolition of the pharyngeal reflex. Lesions of the nucleus of the nerve do not affect the sensation of taste owing to communications of the taste fibers with the trigeminal nerve.

THE PNEUMOGASTRIC NERVE

The pneumogastric, or vagus nerve, arises from a nucleus beneath the floor of the fourth ventricle along with the nucleus of origin of the glossopharyngeal nerve. The nerve has a very extensive distribution, supplying motor fibers to the palate, pharynx, and larynx. It also sends fibers to the esophagus, stomach, heart, lungs, and through the sympathetic system to the intestines and spleen.

Paralysis of the pneumogastric nerve produces unilateral paralysis of the palate. This paralysis is demonstrated by observing the excursion of the palate while the patient pronounces a syllable, such as the word "Ah," when only half of the palate rises in the normal manner. In a patient with palatal paralysis

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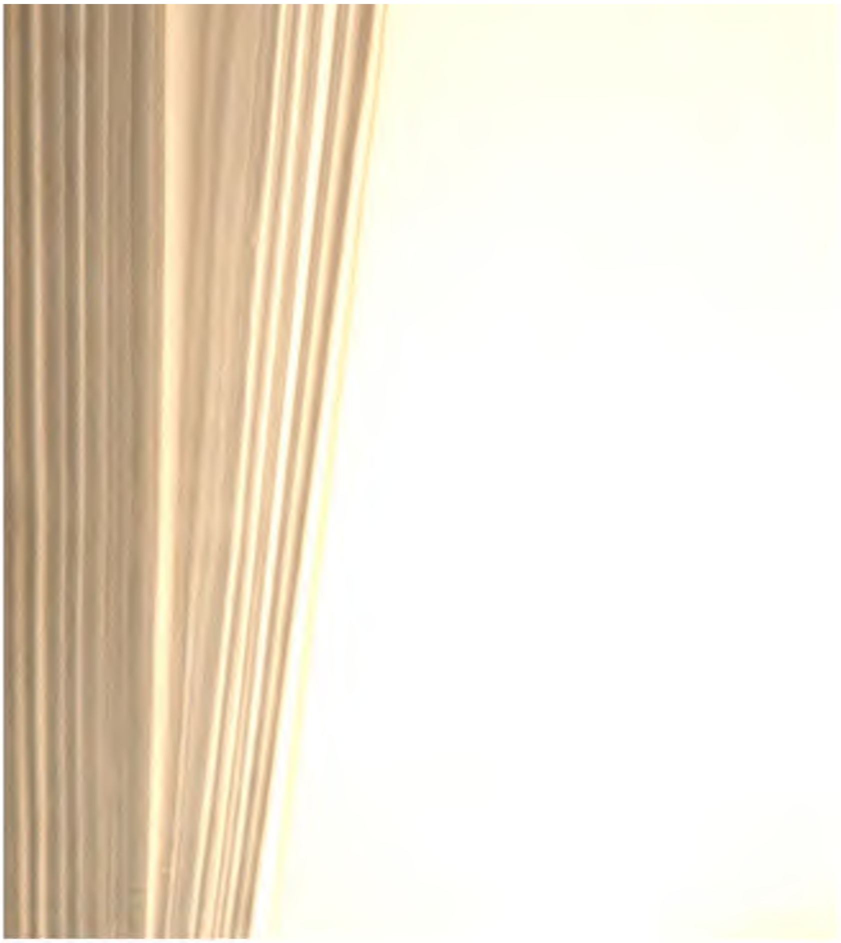

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